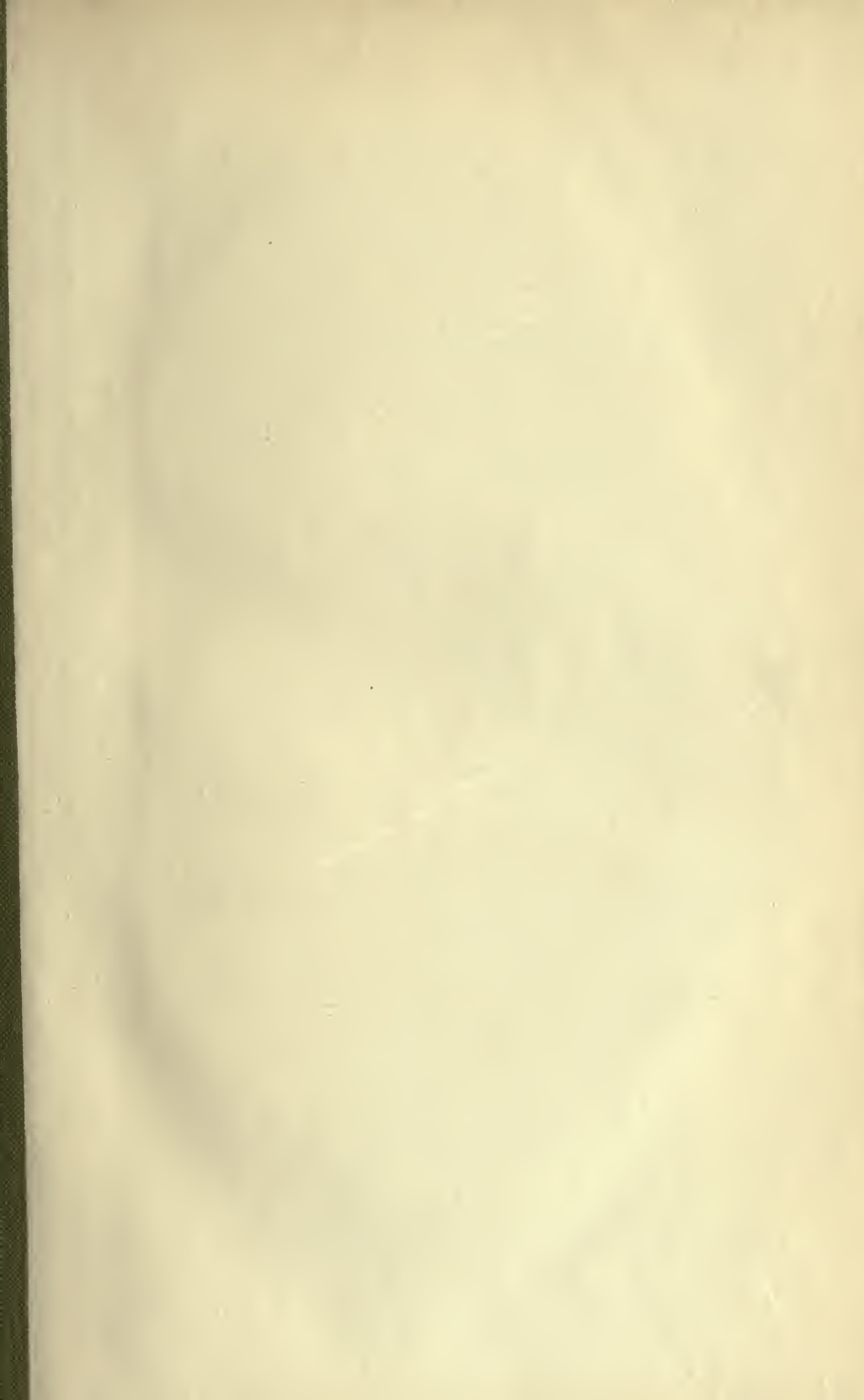


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THE CLINICS

OF

JOHN B. MURPHY, M.D.

AT

MERCY HOSPITAL, CHICAGO

VOLUME III, 1914

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PHILADELPHIA AND LONDON

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NOTE

Many of the cases referred to in this number, in connection with the clinical talks of Dr. John B. Murphy, have already been reported in past numbers of the CLINICS, and the same can be said in regard to the skiagrams and photographs illustrating these cases. These were, however, shown in connection with the clinics held during the Clinical Congress of Surgeons, November 1-15, 1913, and are, therefore, part of the record, which accounts for their repetition. They show, moreover, the further progress of these cases and complete the histories of many of them.

Beginning with the next number (April, 1914) there will appear in each issue of the CLINICS a detailed talk by Dr. John B. Murphy on some special topic connected with the general subject of **Surgical Diagnosis**.

Dr. John B. Murphy's Clinics at Mercy Hospital, Chicago, are held on the following days and hours:

Monday, 9 to 12 For Visiting Doctors and Students
Wednesday, 9 to 1 For Visiting Doctors
Thursday, 9 to 1 For Visiting Doctors
Friday, 10.30 to 12.30. For Visiting Doctors and Students
Saturday, 9 to 1 For Visiting Doctors

All visiting doctors are welcome at these clinics.

FRACTURE OF INTERNAL AND EXTERNAL MALLEOLUS ON A LINE WITH THE TIBIO-ASTRAGALOID ARTIC- ULATION

HISTORY

PATIENT, male, aged forty-two, entered the hospital October 27, 1913, because of an injury to the right ankle sustained the day before. He states that he fell from a ladder to the floor, a distance of eight feet, landing on the inner side of his right foot and turning it out forcibly. Immediately after the injury he began to have severe pain in the ankle, and on examining his foot he noticed that it was turned outward and slightly upward. He had his boy try to straighten it out, and as he did so, the patient heard a clicking noise in the ankle. Then he tried to stand on his foot, but was unable to do so because of the severe pain in and weakness of the ankle. It soon began to swell and became discolored. There are several bruises on the outer side of the ankle now. No injuries were sustained in any other part of the body. He was immediately brought to the hospital.

Examination showed an eversion of the foot, with a downward displacement of the internal malleolus. Palpation elicited a distinct crepitus in the region of the external malleolus. The foot was freely movable in all directions. On attempting inversion a fracture of the internal malleolus was disclosed and the fragment was palpable. The ankle was very much swollen. The skiagram showed a fracture of both malleoli on a line with the tibio-astragaloid articulation. A lateral view showed that the normal relationship between the tibia and astragalus was disturbed—the tibia had slid backward on the astragalus (Figs. 2, 3, 4, and 5).

[Four days later an attempt was made to reduce the fracture, and a plaster-of-Paris cast was applied, extending from the toes to the knee, with the foot at an acute angle flexion and in a line

with the long axis of the leg. The cast was removed on the ninth day, and on further examination Dr. Murphy decided that it was advisable to resort to an extra-articular, non-bloody nailing of the fragments, each to its respective shaft, in order to secure a good anatomic and functional result. This operation was the first one done on the first day of the Clinical Congress, November 10, 1913, and is herein reported.—ED.]

COMMENTS AND OPERATION

DR. MURPHY: Failure to secure a good result from treatment occurs more commonly in connection with a Pott's fracture than with any other type of fracture except a Colles'. Colles' fracture more frequently results in a failure to restore normal conformation, but one not nearly so detrimental to the comfort and service of the patient as is the case with a Pott's fracture. I will show you why. First, however, in order that we may grasp the nature, extent, and purpose of the work to be performed in reducing a Pott's fracture, it is essential that we understand the elements of a Pott's fracture from a practical standpoint.

What is a Pott's fracture? A Pott's fracture is an *eversion* fracture. It is always an *eversion* fracture, never an *inversion* fracture. What do we mean by an *eversion* fracture? It is a fracture of the lower end of the fibula that is produced by the foot turning outward when the injury is sustained. What happens when the foot turns outward? The external surface of the astragalus presses against the tip of the external malleolus, and the fibula breaks primarily. If the interosseous ligament is strong, the fibula breaks within the first $2\frac{1}{2}$ inches above the tip of the external malleolus, otherwise only the tip of the fibula is broken off on a line with the tibio-astragaloid articulation, as in the case before us today. In a typical Pott's fracture the fibula fractures high up.

Next occurs a fracture of the internal malleolus due to traction on the internal lateral ligament or rupture of the ligament. Then, as the pressure is continued with the foot everted and the weight of the patient's body is brought to bear on the foot, what happens? The astragalus is crowded up against the tibiofibular



Fig. 2.—Fracture of internal and external malleolus on a line with the articulation. This view is from behind, and the internal malleolus does not appear to be fractured, although it is not in normal relationship to the shaft. The later skiagrams, taken at a slightly different angle, show that it is fractured off from the shaft. The foot is displaced outward and is inverted. (This and the next skiagram were made the day of the injury.)



Fig. 3.—Lateral view, showing the backward luxation of the tibia on the astragalus. The foot is at an obtuse angle to the leg.



Fig. 4.—Skiagram (anteroposterior view) made six days after the injury, with a plaster-of-Paris cast applied. The fracture of the internal malleolus is now very evident. The lower ends of the fibula and tibia form a straight line. The foot is in the inverted position in which it was held while the cast was being applied. The external malleolus is displaced further inward than in the first skiagram. The lower edge of the tibia dips behind the upper edge of the astragalus, because of the backward luxation of the tibia.



Fig. 5.—Skiagram (lateral view) made six days after the injury, with a plaster-of-Paris cast applied. The foot is nearly at right angles to the leg, but the backward luxation of the tibia has not been overcome. The anterior edge of the articular surface of the tibia strikes against the upper surface of the astragalus, preventing full flexion of the foot.

articulation, and, acting as a wedge, forces apart the tibia and fibula and splits or tears the interosseous ligament. If the force continues still further, anterior luxation of the tibia into the notch on the upper surface of the astragalus occurs, and the foot is not only everted, but hyperextended. The variety of lesions and the amount of deformity depend on the degree of eversion and the degree of force exerted. That is practically all there is to a Pott's fracture, unless the force is continued to the degree of compounding the fracture.

The next proposition to be considered is, how will you manage a Pott's fracture? That depends altogether on how the fracture is produced and what degree of bony and ligamental destruction has occurred and where. There are certain elements in the correction of a Pott's fracture that are vital to a favorable result. What are they?

First: The most dangerous and most serious complication in a Pott's fracture is the anterior luxation of the tibia on the astragalus. How can you ascertain the presence of an anterior luxation of the tibia? The great prominence of the anterior margin of the articular surface of the tibia at the ankle is one point in diagnosis. Another is the backward luxation of the heel, so that the long axis of the tibia falls too far forward on the foot. The foot seems to be foreshortened. The skiagram is another aid in the diagnosis.

Second: If the tendo Achillis is intact and you endeavor to bring the foot up to right angles or better, what happens? You cannot do it on account of the fixation of the tendo Achillis and the forward luxation of the articular end of the tibia, which strikes the upper surface of the astragalus when flexion is attempted. Whenever, therefore, in a Pott's fracture it is impossible to bring the foot to a right or an acute angle with the leg anteriorly, you know you have an anterior luxation of the tibia. That is the proposition which you must bear in mind.

What should you do to relieve the luxation? No amount of traction of the foot forward will pull the anterior margin of the tibia out of its bed in the notch of the astragalus. What can you do? Remember that every fracture is best reduced in inverse

order of the movements in its occurrence. Therefore, inasmuch as this luxation was the last step in the occurrence of this fracture, it is the first step to be overcome in effecting a reduction. Increase the deformity by extending the foot as far as possible, until it is almost on a parallel line with the tibia, then apply downward traction, and make a skid of the upper surface of the astragalus, so that when you flex the foot, the tibia easily slides back into position, and you will know that you have reduced the dislocation when you are able to flex the foot to an acute angle with the leg.

The next proposition is, how will you manage the fracture? Let us see what happens as this fracture occurs. The original accident resulted, first, in a fracture of the fibula; next, a laceration of the interosseous ligament, the integrity of which, as far as the subsequent usefulness of the limb is concerned, is enormously more important to the ultimate result than to secure a union of the fibula. What do I mean? I mean that unless the torn interosseous ligament reunites so as to secure a close contacting of the tibia and fibula, the patient will have a permanent eversion of the foot. In failure of this close union the patient walks for five or six weeks after the accident with his foot fairly flat, but the eversion increases in degree until, after five or six months, he will be walking on his internal malleolus, that is, if there is not an accurate union of the torn interosseous ligament.

How will you insure a union of the interosseous ligament? By securing a perfect contacting of the normally contiguous surfaces of the tibia and fibula and maintaining the contact. How is that accomplished? By reducing the eversion of the foot. With the foot at an acute angle to the leg, adduct it as far as possible, make strong traction on it, and then push it as far to the inner side as you possibly can. Make a forced inversion. What prevents it from going too far? The fibula. An internal displacement is prevented by the pressure of the inner surface of the fibula against the tibia. In the ordinary Pott's fracture you cannot displace the foot too far inward. Retain it in that position, flexed on the leg, and adducted as much as possible. When the foot is not adducted sufficiently, the V-shaped upper surface

of the astragalus is later driven up between the tibia and fibula, separating these bones and preventing close union of the interosseous ligaments. A permanent eversion of the foot, continually increasing in degree, is the result.

In such cases we always put on a plaster-of-Paris-cast, extending from the toes to the knee, making sure that there is no pressure on the external popliteal nerve as it winds around the neck of the fibula, because pressure on this nerve causes a subsequent drop-foot. The cast must be split as soon as it has hardened, to eliminate the possibility of pressure occurring subsequently as the tissues swell beneath the cast.

That is an entirely different course from that we have to follow when there is present a fracture of both the internal and external malleoli, on a level with the articular surface of the tibia. A fracture of both the internal and the external malleolus on a line with the articular surface of the tibia is a dangerous case. Why? Because if you endeavor to dress the foot as far adducted as possible, the upper end of the lower fibular fragment is luxated directly beneath the articular end of the tibia and the internal malleolus is displaced inward farther than it should be. Not only that, but there is an additional luxation—a posterior or anterior luxation of the tibia on the astragalus. That is one of the most complicated and difficult conditions to treat in connection with fractures at the ankle. It cannot be treated properly in any other way than on an operative basis. In no other way are you sure of the degree of replacement of either the external or internal malleolus, and the maintenance of the proper relationship of the tibia with the astragalus.

It is in this condition that we have applied a method of operation which we propose to use in all cases of uncontrollable fracture in the neighborhood of a joint, except those occurring in the senile hip. We have designated this method "the extra-articular bloodless nailing of the fragments." It is a bloodless operation because the only incision which needs to be made is a mere nicking of the skin, an opening just large enough to admit the nail. It is extra-articular because all the work is done without entering the joint and without compounding the fracture,

which is always an element of danger because of the great possibility of having an infection occur later. In other words, you do the least possible operating, and always away from the line of the fracture and the joint, as far as the incision is concerned.

Therefore, in the management of this case we will expose the internal malleolus by a small incision—one not more than a quarter of an inch in length, just large enough for the nail to be inserted. In like manner we will expose the external malleolus. Then drive one nail through the external malleolus up into the shaft of the fibula, and a second nail through the internal malleolus up into the shaft of the tibia. Both of these nails will be extra-articular—outside of the joint. The way may be prepared for these nails by first driving a small drill through each fragment and into its corresponding shaft, or, as I now do, use a *nail drill* and permit it to remain.

That same principle of operating is applicable and indicated in connection with fractures of the lower end of the radius and in fractures near the elbow-joint, shoulder, knee, acromion, etc. If one or both condyles of the humerus are broken off, you can handle the fracture well by this bloodless, extra-articular nailing. You can hold the fragments in apposition during the process of repair without an open operation and without any manipulation of tissues that are bruised and edematous from the trauma of injury.

Will you do that on the day of the accident? *No. The operation is never performed on the day of accident.* All these operations for fractures in the neighborhood of joints are postponed until such a time as the effects of the trauma on the soft tissues have partly subsided. We usually wait from five to ten, eleven, and even twelve days before attempting any operation in these cases. It is well known that a granulating surface is much less easily infected than a freshly lacerated tissue. These internal lacerations are identical with external lacerations, as far as the lowering of resistance to infection and the inflammatory reaction to trauma are concerned; therefore, never perform these operations on the day of the accident. Wait until the tissues have reacted to the trauma, until they will best be able to resist

infection. I speak of infection, because it is the bugbear of all bone and joint surgery. Even with superlative asepsis sterile results are not attained with uniformity.

Now, let us turn to the consideration of the elbow fractures. When the internal condyle is fractured, you have no trouble at all in finding the condyle. As soon as you locate it, nick the skin with a tenotome, drive the drill through the fragment into the shaft, remove the drill, and maintain the fixation with an eight- or ten-penny wire nail. Close the skin incision with one horse-hair stitch, and seal the wound with collodion. That is all. You know the nail is there and there to stay. Absolute immobilization of the fragments is secured, and early passive *painless* motion may be instituted when desired.

If there is a fracture of both condyles, the operation is done in the same manner on the external condyle as on the internal. You do not enter the joint cavity at any time. The tissues are hardly exposed to infection. The arm is placed in a sling. No cast need to be applied, although a posterior wire splint, fashioned from ordinary woven wire, or a light posterior three-fifths plaster-of-Paris cast (either one extending from the fingers to the neck) will give additional security to the bone during the process of repair.

I would be remiss if, in the discussion of this subject, I did not call attention to one of the disastrous practices indulged in by the profession in the management of fractures in the neighborhood of joints, that is, the early institution of *forced* motion. There is nothing that contributes so much to the production of ankylosis in these cases as too early *forced* motion. Keep the limb absolutely immobilized during the process of repair, say, for five or six weeks. If we do that, we will not have an ankylosis occur later.

We have learned two things about fractures: first, *that in cases of fractures in the neighborhood of a joint perfect immobilization practically never results in non-union*, therefore, you may immobilize the limb in the superlative degree; second, *that, in fractures in the shaft of bones, when we immobilize to a superlative degree, there is a tendency to non-union*. In other words, in shaft

fracture cases a small amount of motion at the line of fracture is essential to union. It stimulates osteogenesis.

How much motion we should have is a question. If we secure a good apposition of the fragments in a shaft fracture and then put the patient in ambulation, we will have fewer cases of non-union than we have now. If we resorted to that practice in cases of fracture near a joint, what is the result? We lacerate and relacerate the soft parts and separate the fragments—leading to what? Ankylosis! Our knowledge of the histology of wound repair tells us that an excessive quantity of connective tissue would be produced. That, in turn, is followed by a greater callus or bone formation in the new-formed connective tissue, and the motion of the limb is thereby restricted. Finally, a bony ankylosis may result, the joint being destroyed completely. That is the result of excessive early motion in these cases.

The patient we have here today has had a plaster cast put on in a most careful way. He had an excessive internal luxation, which we believe was detrimental in securing an accurate apposition of the fragments, and we find on examination of the skiagram that there is too much inward luxation if we maintain them in that position. That is why we have decided to operate on him. We are forced to do the operation. We do not operate on a Pott's fracture, as a rule. Operations on Pott's fractures not only are unpleasant, but very difficult. The results that can be obtained in these fractures by operation are well illustrated by the cases which we have had in this clinic.

In this case today there is a posterior luxation of the astragalus on the lower end of [the] tibia. The tibia has slid forward on the upper surface of the astragalus. The foot is thrown backward. If I do not correct this deformity, this patient will be unable to flex his foot to a right angle, and he will be forced to walk on his toes. His foot will be in a talipes equinus position. Just look at the mobility of that foot! I can put it in any position I desire, but it does not remain in place. There is nothing to hold it. The internal malleolus can be moved about freely. The same is true of the external malleolus.

I will place the foot in a position of slight adduction. Why?

FRACTURE OF INTERNAL AND EXTERNAL MALLEOLUS 11

Because the tendency of the foot in a Pott's fracture is always to an eversion of the foot, so it is turned inward beyond the normal anatomic line of the joint. The assistant will hold the foot in this overcorrected or extreme inversion position and flexed to a little more than a right angle while I nail the fragments.

First, I will nick the skin over the tip of the external malleolus with the tenotome. Then I pass a $\frac{1}{16}$ -inch steel drill through the malleolus upward and slightly inward into the shaft of the fibula for a distance of about $1\frac{1}{2}$ inches. The drill is withdrawn, and an eight-penny wire nail is driven into the drill-hole, thus securing absolute fixation of the lower fragment to the shaft. I cut off the head of the nail with a wire nipper because it may press against the skin and be a source of pain, necessitating its removal subsequently. That nail gives us a good apposition of the fragments. A single horsehair suture closes the small skin incision. I have not compounded that fracture—I have not contacted anything, except the drill and the nail, with the line of fracture.

The operation is repeated on the inner side. In attempting to remove the drill it broke—but I do not want any better nail than that is. It is in the full length and securely fixes the fragments in perfect apposition. I will not try to take that drill out. Why should I? It is a more expensive nail than the one we intended to use, but it is not nearly so expensive to the patient as it would be if I were to take it out. Most of the nails we have used in our work have been left in permanently. They do not need to be removed unless they are the source of pain to the patient through pressure on the skin. In that case nick the skin and withdraw the nail. This is not done until sufficient time has elapsed to insure practical completion of the process of repair. A nail with a drill tip has been devised by us. These nails are placed and left in, just squaring the head.

Note how the foot remains in position now without any support. Why? Because we have restored its normal anatomic supports by nailing the malleoli back in place. The foot is dressed with a plain gauze dressing. A plaster-of-Paris cast (Fig. 6) is put on, extending from the toes to the knee.

You can see what a very simple operation that is—how little we have exposed the tissues to infection and how firmly we have secured these fragments. The result is a complete success. The proposition is, how secure are the fragments? This foot cannot be displaced backward or forward. We are sure of the final fair result in this case.

In this connection let me cite a case in which the patient's physician made an error in diagnosis. He knew that the man had a fracture near the ankle-joint and he called it a "Pott's fracture," which it was not. Let me give you the story, because this story makes the diagnosis always. The man was stepping off a platform, and when he placed his foot on the ground, it turned inward and he had a fracture. His doctor made an error in his diagnosis when he called it a Pott's fracture, because, as I stated before, a Pott's fracture never results from an inversion of the foot. It is always an eversion fracture. An inversion of the foot may result in a fracture of the lower end of the tibia and fibula, but not of the Pott's type.

The doctor put on a cast, and when he removed it five weeks afterward the foot was in splendid position. The patient was permitted to walk on it. After eight weeks it began to turn in, and it continued to do so. When he came to us his external malleolus was on the floor, and the foot was turned in, not out (Figs. 7-14).

What question should you always ask such patients? "How much did you suffer when you had the injury?" This man said, "Why, do you know, doctor, I never had any pain, even when I broke it." That tells the story. That is always the story of a tabetic. There is no pain with the fracture, or practically no pain. The skiagram showed that he had a fracture of both malleoli and an absorption of the lower ends of the tibia. The foot was turned in, but he had no pain, either when he was injured or afterward. He had the Argyll-Robertson pupil, and a positive XXX Wassermann, and other luetic manifestations, which showed that all his trouble was the sequence of his lues, and not the sequence of his doctor's management, which is exactly in consonance with a case of that kind which we will show you



Fig. 6.—Full-length plaster-of-Paris cast after it has been split with the Gigli-saw.



Fig. 7.—Charcot ankle. The foot is displaced inward and the external malleolus almost touches the ground.



Fig. 8.—Lateral view of foot, showing how the external malleolus has dropped down and the destruction of the normal conformation of the ankle. It is thickened and pushed downward onto the foot.



Fig. 9.—Lateral view of foot from inner side, apparently normal except for the thickening of the ankle.



Fig. 10.—Ankle viewed from behind forward. The foot is displaced inward. The lower end of the tibia, the internal malleolus, and the upper surface of the astragalus are completely destroyed.



Fig. 11.—View of foot from in front backward. The destruction of the astragalus shows more clearly than in the previous picture, with a great internal displacement of the foot.



Fig. 12.—Lateral view of foot from inner side, showing how the tibia has apparently been driven down into the astragalus. The lower end of the tibia is also greatly thickened and displaced backward, as well as downward.



Fig. 13.—Foot viewed from behind after operation, showing how the foot was placed under the tibia in practically its normal relationship, and how fixation was secured by means of three wire nails. One large nail was driven up through the sole of the foot, through the os calcis and astragalus, and into the tibia. The fibula was fractured to permit of an approximation of the shortened tibia with the astragalus, and the lower fibula fragment was fixed to the tibia by means of two wire nails. The head was removed from one of the nails. These nails kept the foot securely in position until the tibia had united with the astragalus.



Fig. 14.—Lateral view of foot after operation. The position of the foot is good. The tibia rests squarely on the top of the astragalus, and the long wire nail maintains the foot in a right-angle position to the leg and prevents its displacement. The two smaller nails pass through the fibula into the tibia, as shown in Fig. 13.

this week. That man has the Argyll-Robertson pupil, the absence of the patellar reflexes, and the positive Romberg. He presents the entire picture of tabes. I have had these cases with no other symptoms than the Argyll-Robertson pupil still show the typical tabetic joint after fracture.

In one of our cases a man sustained a fracture of the hip when he fell off his bicycle. He was sent to me some weeks afterward with a diagnosis of sarcoma of the femur. On questioning him, he told me that after his fall he got back on his bicycle and rode home, and that he worked for six weeks afterward. At no time did he have any pain in his hip.

The skiagram showed that the neck of the femur had been fractured, and that an enormous callus had formed, extending as high as the crest of the ilium. It was a mass of bone as large as my hat (Figs. 15-20). The absence of pain proved conclusively that it was not a sarcoma, and that the fracture was of tabetic origin. On examination every evidence of lues was elicited. A sarcoma is never a sequence of a fracture. Sarcoma is a sequence of a milder injury to a bone.

VISITING PHYSICIAN: How do you treat a compound fracture?

DR. MURPHY: How do we manage compound fractures primarily? Not by nailing, plating, wiring, or the use of other devices suggested for effecting immobilization of a fracture. *No, absolutely no!* If you attempt to do that, exposing and handling compound fractures, you invite infection and insure serious complications in a large percentage of the cases. If, on the other hand, you attempt to secure as good an apposition of the soft parts as you can, and maintain them absolutely quiet during their repair, you can eventually resort to such operative procedures as may be necessary to effect union of the fragments without any hazard to the patient at all.

The proper management of a compound fracture is to keep everything out of the wound from the time the fracture occurs. Do not touch it with your hands. Do not touch it even with the gloved hand. Touch it as little as possible with instruments. Place the soft parts in as good apposition as they can be placed after you have covered the injured surface with iodine; then dress

the limb in the best immobilizing position that you can. *Do not scrape, do not wash, do not handle!* A large percentage of these cases so managed will go on to a complete primary healing, many without a secondary operation for correction of bone displacement.

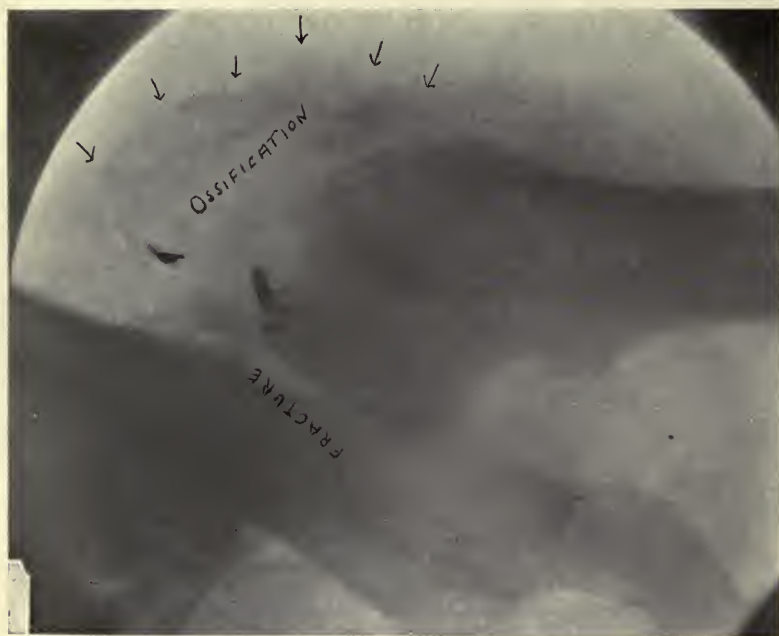
Here is a case [indicating a patient] illustrating the management of this class of case. This patient left the hospital about a week ago. He came here in June with a compound fracture of the tibia and fibula at about the junction of the lower and middle thirds and an overriding of the fragments of about two inches. We endeavored to reduce the deformity by traction and external manipulation, but failed. We dressed the wound and waited for it to heal. He remained quiet from June until October, but with the deformity present all the time. Toward the end of September the wound was all healed (Figs. 21, 22, 23).

On the first day of October we made an incision on the front of the leg, just to the outer side of the tibia, and over the seat of the fracture, and freed the ends of the fragments of soft tissue and callus, so that we had raw bone surfaces before attempting to secure apposition of the fragments. Then we effected an apposition by means of our special bone lever (Figs. 24, 25). We inserted one end of the lever between the two fragments, and by making a steady pull on the other end, a good apposition was easily secured. No plate, no wires, no nails were used—merely a plaster-of-Paris cast, and the fracture has healed perfectly, without deformity and without hazard to the patient.

The plaster-of-Paris cast has been applied in the case just operated. It was put on as a circular cast, but we never let a circular cast go out of the operating-room without dividing it, so that if it causes pain or if the patient has pressure from it, he can open the cast himself and get relief. A Gigli saw is laid on the limb lengthwise and the plaster is put on over it. When the plaster has set, grasp both ends of the saw, pull it forward and back, and the cast is cut. It was first used by Dr. Austen, of Indiana, who was my intern in the Alexian Brothers Hospital. I thought it was my idea, but he reminded me one day that he had used it some two years before.



Figs. 15, 16, and 17.—In these photographs the mass over the left hip shows clearly. It appears to be located higher than the upper extremity of the femur, and extend clear up to the upper margin of the ilium. The resemblance to sarcoma is striking.



Figs. 18, 19, and 20.—Skiagrams of Charcot hip taken at different angles, showing a fracture of the neck of the femur, and an enormous ossification of the fibrous layer of the capsule, extending from near the base of the trochanter outward, upward, and then inward toward the ilium.



Fig. 20.—For description see Figs. 18 and 19.



Fig. 21.—Compound fracture of tibia and fibula, with displacement of the fragments and absence of bony contacting. No union (anteroposterior view).



Fig. 22.—Lateral view of compound fracture of the tibia and fibula. The displacement of the fragments is apparently not as great as in the previous picture.



Fig. 23.—Skiagram (anteroposterior view) made eighteen weeks after the first two skiagrams and two weeks after the tibia fragments had been exposed and placed in their present position by means of Dr. Murphy's bone skid. The apposition and position of the tibia fragments are perfect. No effort was made to secure apposition of the fibula fragments.



Figs. 24 and 25.—Dr. Murphy's lever and bone skid, viewed from both sides. This lever is used to secure apposition of displaced or overriding fragments in fracture and to effect a reposition of the head of the femur and humerus in their respective sockets after dislocation.

Let the record show that the external malleolus was first exposed by a puncture of the skin with a tenotome and the drill then passed through the malleolus, up into the lower end of the tibia, and an eight-penny nail without a head inserted.

Let the record further show that on the internal side the malleolus was exposed in a similar manner and the drill was then used; when the drill was in to the full extent, it broke off at its base; and that it was permitted to remain in position, as it would fulfil the same purpose as a nail. Each skin puncture was closed with a single horsehair stitch. Union per primam.

ANKYLOSIS OF HIP DUE TO "LIPPING" OF THE RIM OF THE ACETABULUM; A COLLAR OF BONE ON THE NECK OF THE FEMUR; CHEILO TOMY; ARTHROPLASTY

HISTORY

PATIENT, female, aged thirty-nine, entered the hospital November 8, 1913, because of a constant, dull pain in the left hip, which is exaggerated on walking or jarring the leg, and with great limitation of the range of motion in the joint. The present trouble dates back fourteen years, when the patient gave birth to a child. It was a forceps delivery. The child's neck was broken and it was born dead. The placenta was not removed. On the fifth day after delivery there was noted a very foul discharge from the vagina. The next day the patient had a chill, with a temperature of 105° – 106° F., and soon afterward she became delirious. A consultant was called on the ninth day. He curetted the uterus (in the patient's home), and found and removed the placenta. A few days later she began to have *severe* pain in the left hip. It was very sensitive to touch, and the slightest movement increased the pain. This was thought to be "rheumatic." An extension was applied to the leg some time during the second week, with 25 pounds weight attached to it. After two months of this treatment at home she was taken to a hospital, where she remained for another two months. The extension was left on the leg all this time, but the weight was gradually reduced.

At the end of two months the extension was removed, and she was allowed to be up and around on crutches, but she did not then try to put any weight on the left leg or even move it, because of pain when she attempted to do so. She used the crutches

all the next year, and during that time she gradually began to put weight on the leg and to move it, but she had constant dull pain in the hip, which was increased in severity on walking or jarring. This condition of affairs kept gradually getting worse until four years ago (1909), when she noticed that the motion in the hip was getting less and the pain was becoming more severe. Since last May (1913) this pain has been very severe, and motion has been almost entirely abolished. She cannot lie on her left side because of the pain in the hip, but even when lying on her right side, the pain is almost as severe. She has been taking baths (hydrotherapy) off and on since the onset of this trouble, because she was told that she had "rheumatism." She does not remember ever having had any injury to the hip. A skiagram was not made until four days ago.

COMMENTS AND OPERATION

DR. MURPHY (November 15, 1913): When you look at that skiagram, you will at first glance say that this is a bony ankylosis of the hip. I do not think so. It looks to me, and I think the history bears me out, as though this woman had a chronic synovitis and that this mass of bone on the rim of the acetabulum is new-formed bone, the result of ossification of the white fibrous tissue of the capsule. It is exactly what is apt to occur in connection with a chronic peri-articular inflammation of infectious origin. It follows exactly along embryologic lines. The fibrous portion of the capsule is merely a continuation of the embryologic periosteum before the joint had formed. It appears to me that this woman has had a very severe synovitis. She has been an invalid for fourteen years (Fig. 26).

Let us review this history. It is fairly typical of this class of cases. The pain in the hip was not "rheumatism." It was the sequence of a metastatic infection in her hip. She had a puerperal infection, with chills and fever, and this was followed by a metastatic infection in the hip. This metastasis was initiated by a chill, which was indicative of a severe type of infection and meant that, in all probability, it would cause a destruction of the joint unless the case was well managed—and it was.



Fig. 26.—Apparently a complete bony ankylosis of the hip, before operation. The normal anatomy of the joint has been destroyed; the femur and acetabulum appear to form a solid bony mass. At operation the joint cavity was deeper and the cartilage eroded and the lipping caused the apparent ankylosis. A collar of bone had formed around the head of the femur, and there was a deposit of bone on the margin of the acetabulum—the so-called “lipping.”

She had an extension on that leg for four months, and as a result of that treatment she has a straight limb, and, I think, normal articular surfaces, because the ankylosis is peri-articular. The most favorable conditions are present for making a new joint. We believe, however, that she has a joint there now, in spite of the fact that the skiagram does not show it.

What are we going to do in this case? We are going to perform a typical arthroplasty, first, however, removing this ridge of bone on the margin of the acetabulum and the collar of bone around the neck of the femur. The pedicled flap of fat and fascia lata, which we will place between the head of the femur and the acetabulum, will effectually prevent the recurrence of this lipping. I will explain the steps of the operation as we proceed.

This will be a typical arthroplasty, such as we perform in cases of bony ankylosis of the hip-joint, even though the pathology present here is not that of an intra-articular bony ankylosis. As I said before, the skiagram would suggest that there was such an ankylosis present, but having examined it very carefully, I have come to the conclusion that the ankylosis in this case is due to a locking of the joint caused by the production of new bone on the edge of the acetabulum,—the so-called lipping,—and the collar of bone which encircles the neck of the femur. I do not believe that I am in error in my interpretation of this skiagram and analysis of this case.

The patient is now ready for operation. We always use nitrous-oxid-oxygen-ether anesthesia, and our results have been so good that we see no reason why we should change our method. Sister Ethelrida is an experienced anesthetist, having given many thousand, and we are perfectly comfortable when she is giving the anesthetic. The patient is placed on the right side, so as to give us good access to the left hip. The skin has been sterilized with 3.5 per cent. tincture of iodine, the strength we always use in this clinic, because we have found that in a number of instances the 7 per cent. tincture, which is the full strength pharmacopeial tincture, has caused a dermatitis. You will notice that one of my assistants has charge of this leg, so that any manipulation which I may wish to have made will be made

promptly and effectually. Having placed the sterile towels and sterile sheet so as to give us a good exposure, and having encased the leg in a sterile stocking extending up to the knee, we are ready to make the incision.

I have made a number of incisions in these cases according to what I thought would give me the best exposure, but I have found that the best incision is the so-called "U"-shaped incision—one which allows us to secure a good flap for interposition between the acetabulum and the head of the femur. The incision is begun about one inch below and to the inner side of the antero-superior iliac spine. It passes downward, outward, and upward so that the trochanter is located beneath, in the middle of the flap, the incision ending about five inches behind the starting-point. It extends down through the skin and fat onto the aponeurosis of the muscles—the fascia lata. This patient is rather stout, so that the flap will be a thick one. However, I would rather do an arthroplasty on a fat patient than on a thin patient, because we get much better results in these cases when we can secure a fairly fat flap. In these hip cases the interposing flap is always taken from the fascia lata and fat above it. The fat is an advantage in the operation, but it is certainly a disadvantage for demonstration. This skin and fascia flap is now retracted upward.

The next step is the removal of the great trochanter with its attached muscles. This is done by passing the chain saw underneath this group of muscles from behind forward, and then sawing downward and outward, so as to remove the trochanter near its base. Remember that the muscles attached to the trochanter are not disturbed, so that when the trochanter is subsequently replaced, we retain the full action of these muscles. This is essential to a successful outcome in these cases.

You can see now what a considerable exposure that gives us about the hip-joint. The capsule of the joint is very thick, and we will divide it with the scalpel. As we enter the joint cavity the joint fluid flows out, showing that we are in the right zone. The "lipping," to which I called your attention before commencing the operation, now shows up very well. It is particularly well

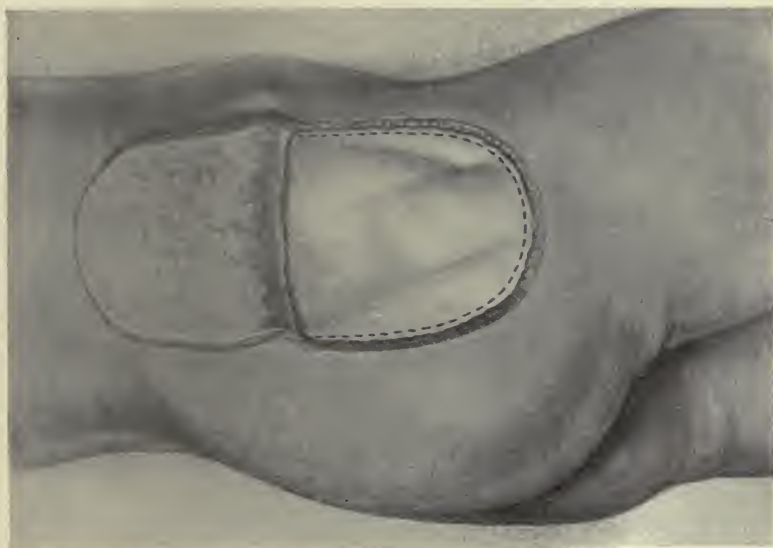


Fig. 27.—Essential steps in Dr. Murphy's hip arthroplasty as depicted in the *Lyon Chirurgical* (September, 1913) by Drs. Guillot and Dehelly. U-shaped incision with skin-flap elevated and trochanter major showing through fascia lata.



Fig. 28.—All fascial structures have been retracted upward: the trochanter and its attached muscles are in view; the chain-saw has been passed underneath this muscle group, and the trochanter is being sawed off. The dotted line on the femur indicates the direction of the trochanteric division.



Fig. 20.—The trochanter with its attached muscles is drawn upward, the anterior fibers of the gluteus medius have been cut, and the capsule of the joint is being incised.

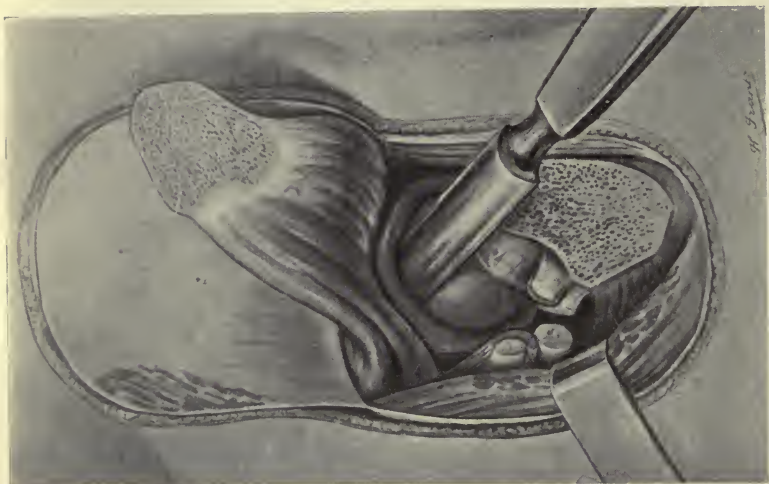


Fig. 30.—The large curved chisel is being driven between the head of the femur and the acetabulum to separate the ankylosis.

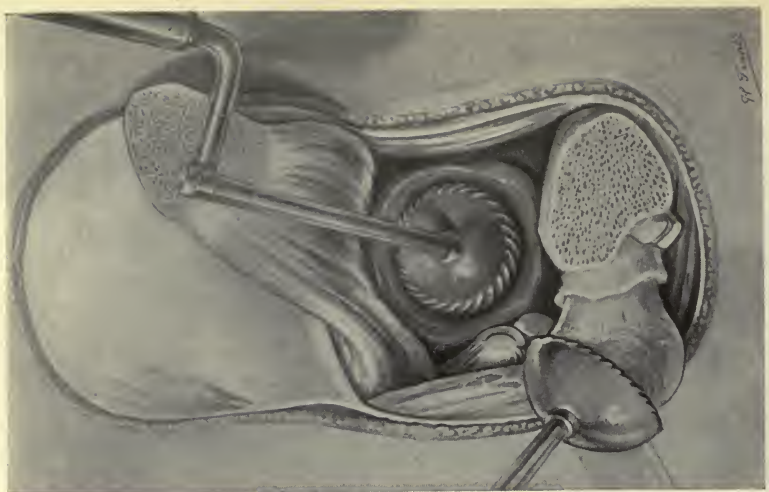


Fig. 31.—The head of the femur has been dislocated out of the acetabulum, and it is being shaped with the Murphy end mill. The Murphy reamer is in the acetabulum, and is shaping the latter to fit the new femoral head.

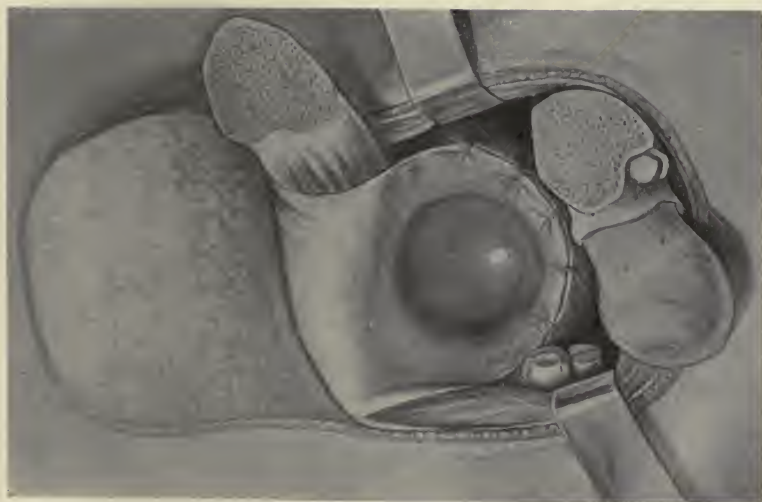


Fig. 32.—The interposing fat and fascia flap has been dissected free from the under surface of the skin-flap, dropped down over the acetabulum, and sutured to its anterior, posterior, and lower margins with chromicized catgut. The head of the femur, when replaced, will rest on this flap.

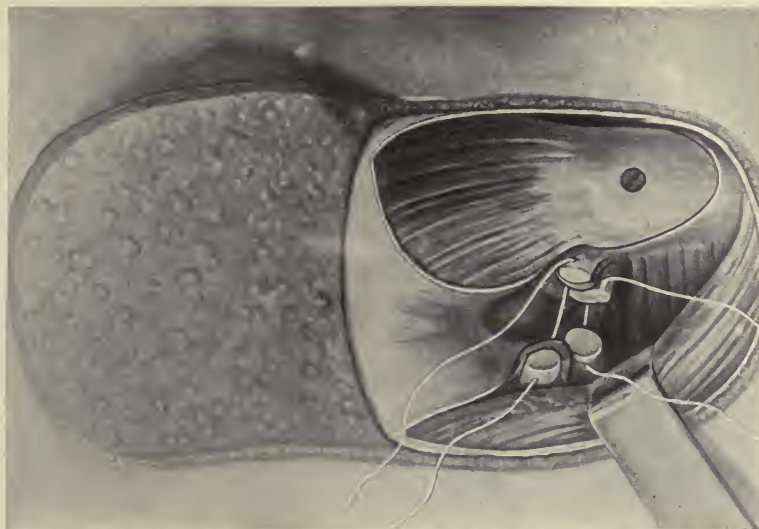


Fig. 33.—Trochanter nailed back in place and sutures in ends of cut pyriformis and obturator externus muscles, ready for tying. The interposing flap passes down in front of the muscles attached to the trochanter.

developed on the anterior margin of the acetabulum. As the leg is rotated slowly outward you can see distinctly the collar of bone on the head of the femur, just about where it joins with the neck. There is no intra-articular ankylosis; that is, the head of the femur has not united to the acetabulum. It is a very beautiful picture, and the condition present confirms the diagnosis which we made before the operation, even though the skiagram apparently showed a bony ankylosis.

The motion in the joint was prevented by this lipping and the collar of bone, and that is why this patient had pain. If she had had a bony ankylosis, she would have been free from pain. The operation which I will do here in combination with the arthroplasty was recently described in one of the British journals as a "cheilotomy."

In order to prevent the reformation of bone on the acetabulum and the head of the femur, we must interpose a flap of fat and fascia lata, just as we do in the typical arthroplasty for a bony ankylosis without the lipping. By flexing the leg and making a strong external rotation, the head of the femur is easily thrown out of the acetabulum. It is not necessary to use the chisels to separate the ankylosis here, because there is none, but we will remove this lip of bone from the margin of the acetabulum and the collar of bone from the head of the femur with the curved chisel, and then, in order to have a normal conformation of this joint, we will ream out the acetabulum and shape the head of the femur with the end-mill, proceeding in exactly the same manner as we would in performing our usual arthroplasty on the hip [removes lip of bone].

We have removed the lip of bone on this acetabular rim. It was nearly two inches deep, and consisted of dense cicatricial bone, as was made evident by the difficulty with which the chisel could be driven into it. The greatest thickening here was on the anterior and upper surfaces, so that abduction was absolutely impossible. This is a much more difficult operation than the usual arthroplasty, which can be done in one-third the time that it will take to do this operation. If I do not remove that excess of bone, the patient will not get any relief from the operation.

If I do not interpose a flap of fat and fascia lata, the condition will recur.

Having removed this excessive bone on the acetabular margin and head of the femur, and having restored the normal anatomy of the joint as nearly as it was possible to do, the next step is to prepare and place the interposing flap. This flap consists of fascia lata and fat, and is dissected free from the under surface of the original skin and fascia flap, which has been retracted upward all this time. It is about one-half an inch in thickness and of the same shape as the skin-flap, but nearly twice as broad and large, the pedicle being at the upper end, so that the circulation of the flap will be insured. You can see how easily this flap can be brought down so as to cover all the acetabulum, and when the head of the femur is replaced in its normal position, this flap will be interposed between it and the acetabulum, covering all the raw, bony surfaces, so that the new bone formation cannot recur, and ankylosis will be obviated. The muscles, which are attached to the trochanter and which have also been retracted upward, are behind this flap; that is, when the flap is dissected free from the skin-flap it is dropped down in front of the trochanter and its attached muscles. That is the natural position for it to take. It is not necessary to go under the muscles in order to place the flap; it falls naturally into its position, and it forms the new lining of the hip-joint. This flap is fixed by a few tacking sutures of catgut to the margin of the acetabulum in front and to the neck of the femur behind and below, thus completely investing the head of that bone when it is replaced in the acetabulum. We use chromicized gut for these tacking sutures, and not more than three or four need be placed to secure a good position of the flap.

We are now ready to replace the head of the femur in the acetabulum, and we accomplish this by rotating the leg inward, then straightening and abducting it, carrying the knee through a half-circle. You will note that the head comes into position very easily, and that it remains in position. We have not attached the old capsule, because restoration of the capsule is not essential to the integrity of this joint. As we have said before, the capsule of the joint is called into play as a support only when the normal

range of motion is exceeded, and in these cases we believe—and that belief is based on the observation of a large number of cases—that a new capsule is formed from the connective tissue around the joint.

Our patients have had a splendid return of motion in the hip after an arthroplasty, not “nearly” normal, but as good as normal, and at no time has there been any dislocation; therefore, we pay no heed to the capsule.

The trochanter will now be brought down and placed in its normal position on the femur. It will be fixed by means of one wire nail driven through up into the neck of the femur. The muscles attached to this trochanter will hold the head of the femur in contact with the acetabulum and prevent luxation. The deep tissues are next approximated with chromicized catgut, so as to give additional security to the joint, and to close the defect in the fascia lata. The skin is closed with horsehair and a few silkworm-gut tension sutures. You will note there was very little bleeding here—so little, in fact, that it was not necessary to resort to the use of ligatures. Whenever there is any bleeding we always ligate, or, better, use the angiotribe (Blunk), because we fear a hematoma in these cases as much as we do infection. You may have observed that during the entire operation we never touched the wound with the gloved hand; only absolutely sterile instruments contacted with the wound. That is one of the great practices which make for success in this operation, namely, to observe the strictest possible asepsis.

The wound will be dusted with bismuth subiodid, the desiccating powder which we always use, and sealed with collodion gauze. We do not insert a drain. The leg will be dressed in an abducted position with a Buck’s extension applied, and attaching to it a 20-pound weight. The stitches will probably be removed in about two weeks, and the patient will be allowed to sit up in bed after about three weeks, in a chair after about four weeks, and if she does as well as the hips previously operated on have been doing, she will be around on crutches in about five weeks. These patients usually leave the hospital in from seven to ten weeks, and in no instance have we failed to restore a good range of motion in the hip (Fig. 34).

The weight is placed on the leg for the purpose of keeping these joint surfaces separated sufficiently so as not to interfere with the vitality of the interposing flap. The natural tendency of the muscles is to contract, and by doing so, the articular surfaces of the bones are brought into apposition, and if a flap of fat and fascia is interposed between them, the result would be a pressure necrosis of this flap, which would interfere with the successful outcome of the operation; therefore we keep these joint surfaces separated by means of a Buck's extension. After the patient is up and around and making some use of the hip, it is no longer necessary to put the weight on the leg, except at night, because the contraction of the muscles is counteracted during the day by the motion of the joint.

Let me show you a few of these cases. Here is a patient [presenting patient] for whom we performed an arthroplasty on the left hip four weeks ago for a complete bony ankylosis. She also has a bony ankylosis of the right knee. A metastatic infection from a tonsillitis was the cause of the ankylosis. The metastasis was *initiated by a chill*, and that nearly always means that a bony ankylosis will take place in the joint involved. When the metastasis into the joint is not initiated or accompanied by a chill, a bony ankylosis is not likely to occur. Fortunately, this patient's limbs were in good position; they were straight when the bony ankylosis occurred. We had little difficulty in performing the operation. We have not operated on the knee—only on the hip. She has been in a wheel-chair for a week. We will now ask her to stand [the patient is assisted to her feet]. Although the hip is still weak, you will note that she stands well and she has good motion in the hip—almost the normal range of motion only four weeks after the operation. That is less time than is required for the healing of a fracture. She is going to have a splendid hip, and will probably be able to leave the hospital in two or three weeks. She has no pain in her hip when the joint is put through the normal range of motion. (See Figs. 35 and 36.)

VISITING DOCTOR: Has a luxation of one of these hips ever occurred after an arthroplasty?

DR. MURPHY: No. Let me show you a picture of another



Fig. 34.—Skiagram made two months after the operation. The conformation of the joint is good. The nail is in the trochanter. By comparing with the skiagram made before operation, it will be noted how much bone was removed from the head and the rim of the acetabulum.



Fig. 35.—Complete bony ankylosis of the hip-joint before operation. The femur appears to be smelted into the acetabulum, with some upward luxation.



Fig. 36.—Skiagram made five weeks after operation. Although the outlines of the joint are very indistinct, the patient had a normal range of motion, and was able to stand and bear her full weight on the leg. Even at this early date, with only slight support under one arm, the patient walked across the floor.

very interesting case of ankylosis of the hip in which a splendid result was obtained by an arthroplasty. The patient in question had repeated attacks of tonsillitis, fever, nausea, vomiting, and jaundice, but the illness which was directly responsible for the ankylosis of the hip occurred about three years before we saw the patient. She had an attack of influenza, but not of sufficient severity to confine her to bed. On the eleventh day after the onset of the illness, when she was nearly well, but while a purulent rhinitis and bronchitis were still present, she carried an ice-cream freezer on her left hip a distance of 300 yards. She remembers distinctly that the weight of the freezer caused some pain in her hip, and that when she placed it on the ground, the muscles on the left thigh were sore. The following morning she felt chilly and her whole body ached. Gradually the pain apparently localized in the left thigh and hip. Three hours later her temperature was 102° F. and the pain in her thigh and hip was so severe that she was given morphin. In order to get some relief from the tension she flexed the thigh slightly on the abdomen and rotated it inward, adducting the leg. She was confined to her bed for a number of months, the pain in her hip-joint being excruciating all the time.

Not until four weeks after the onset of the trouble was a Buck's extension put on the leg. It was left on for about three weeks, but gave her only little relief from the pain. The extension was then removed, and she was allowed to sit up in a chair, but the pain in the hip was there all the time. She finally got around with the aid of crutches, and the pain in the hip gradually subsided, until it finally was elicited only by putting weight on the leg. Then it disappeared entirely, as did also the motion in her hip. When she had no more pain her hip was stiff—absolutely stiff. When we first saw her her leg was rotated inward and adducted across and beyond the pelvic inlet. There was no motion in the hip. The skiagram showed a bony ankylosis between the femur and the acetabulum.

This was a typical case of metastatic infection in a joint, the result of the influenza, and precipitated in the hip-joint by the trauma incident to carrying the ice-cream freezer on her hip. The history in these cases always tell the same story. We per-

formed the usual arthroplasty in this case, with the usual good result, although the patient was very intractable. Nine months after the operation we received a letter from her stating that she had such good motion in the joint that she could not realize that it had ever been stiff. The pictures corroborate the statement (Figs. 36A and 36B).

Let me cite briefly another case of ankylosis of the hip in which we performed an arthroplasty eighteen years after the ankylosis occurred. The patient told us that she caught cold, and that two weeks later she had pain and soreness in the region of the right hip. The pain increased in severity, and on the evening of that day it was excruciating and apparently localized in the hip-joint. The following morning she said she had a high fever, which continued for several weeks. Motion in her hip caused pain, but apparently was normal in range. After two weeks the patient suffered a nervous breakdown. She was ill for four months, and remembered little of what happened during that time, but when she finally got well, her right hip-joint was stiff and it remained so for eighteen years (Figs. 37-41).

When we examined her we found, both on physical examination and in the skiagram, that she had a bony ankylosis of the hip-joint, which, however, involved only the lower two-thirds of the articular surface of the head of the femur. We performed the usual arthroplasty, and the case progressed uneventfully. The patient left the hospital seven weeks after the operation, with a full range of motion in the hip and was able to bear her weight on that leg. Unless one knew which hip had been operated on, it could not be detected; in fact, the motion in the operated hip was better than that in the fellow hip. She could kick the door-knob in four weeks. Of course, she was the right kind of a patient; she was willing to stand a little pain because she wanted a good hip. Our experience has been that women will work hard and tolerate pain to get results and that men will not. Men do not stand pain as women do. The arthroplasties which we have done on male patients with few exceptions have not resulted nearly so well as have the same operations done on female patients. I think that is the general experience of all surgeons with all kinds of work.



Fig. 36A.—Complete bony ankylosis of left hip with rotation of leg inward and adduction beyond pelvic inlet.



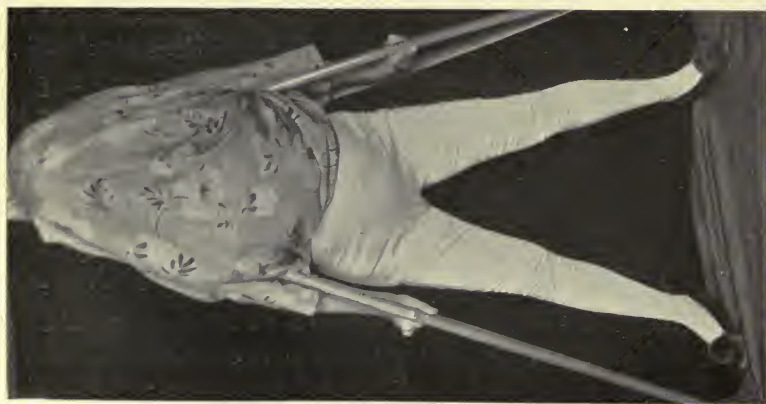
Fig. 36B.—Photographs made one year after operation, showing that patient has a full normal range of motion in the hip on which the arthroplasty was done.



Fig. 37.—Ankylosis of right hip of eighteen years' duration. The upper two-thirds of the head of the femur were firmly united with the acetabulum by a bony ankylosis; in the lower third the ankylosis was fibrous.



Fig. 38.—Skiagram made six weeks after operation. The joint cavity shows clearly. The leg was turned inward when this skiagram was made, hence the conformation of the femur does not appear as usual. The nail is driven through the trochanter into the shaft.



Figs. 39, 40, and 41.—Photographs taken seven weeks after operation, showing the splendid range of motion in the hip. The patient could sit comfortably, stand squarely on the leg, and abduct it very well.

COMPLETE BONY ANKYLOSIS BETWEEN TIBIA AND PATELLA AND FEMUR; AR- THROPLASTY; ACUTE METASTATIC ARTHRITIS

HISTORY

PATIENT, female, aged twenty-eight, entered the hospital November 8, 1913, because of a complete ankylosis of the left knee and considerable limitation of motion in the right.

The present trouble dates back to January, 1908, when the patient was just recovering from what was diagnosed as chicken-pox. She exposed herself to dampness, and soon afterward had a chill and severe pain in the left knee. She thinks she had some elevation of temperature. She went to bed at once. The pain soon became very severe, and she noticed that the knee was swollen. At this time she also had a yellowish discharge from the vagina, and she was told by her doctor that the inflammation in her knee was probably due to the same trouble that caused the vaginal discharge. No bacteriologic examination was made of the discharge. The pain became so severe that in order to get relief she flexed the leg at the knee and had a pillow placed under the knee. She was given an ointment to apply locally and morphin hypodermically, as often as every two hours, to relieve her from the pain. About the third week she had a severe pain in the right knee, and this soon began to swell. She kept both limbs perfectly quiet now, because of the pain caused on attempted motion.

She remained in bed eight months. The pain and swelling had greatly lessened in severity and degree, but at this time she noticed that both knees were stiff. She was taken to a hospital, where she remained four months, and during all this time ointment was applied to both limbs. After this she got up on crutches

but was unable to move her knee-joints. The right leg was straight, but the left one was slightly flexed at the knee. For the next eleven months she was treated by an osteopath, who massaged the limbs, but the condition did not improve. No other joints were involved. Her personal history is otherwise negative.

The skiagram (Fig. 41A) shows a complete bony ankylosis between the femur and tibia, and between the patella and femur, with a subluxation of the tibia, and flexion of the joint to an acute angle.

COMMENTS AND OPERATION

DR. MURPHY (Operation, November 13, 1913): What is wrong with that history? It does not give the date on which the knee involvement began compared with the onset of the chicken-pox. That is a serious mistake. Why? Because you never have a metastatic arthritis in the early stage of any of the exanthematous diseases except scarlet fever. That is a streptococcus infection and the microorganisms may get into the blood in the early stage of the disease and produce metastases. In the other exanthematous diseases metastasis occurs usually in the recrudescence of the disease. The infection is carried into the joints from the pustules on the surface of the body. We have all seen cases of suppuration of the skin, such as furuncles, carbuncles, and pustules, in which a typical metastatic infection into a joint took place. The intern should have put in the day of the chicken-pox on which the joint metastasis occurred, then we could establish a direct relationship between the chicken-pox and the arthritis, because there are definite times when these metastases take place. From the full and correct history, giving all the dates, you can easily trace one back to the other.

All metastatic joint infections that are initiated with a chill are not of the "rheumatic" type. Why do I say that? When I was a student in the late seventies, the late Dr. J. H. Hollister, of this city, who gave the only lecture on pathology I ever had, said, that when a patient had an inflammation in a joint which was initiated by a chill, that meant pyemia, and when he had an inflammation in a joint not initiated by a chill, it was "rheu-



Fig. 41A.—Ankylosis of knee: Complete bony ankylosis between tibia and femur and patella and femur with knee flexed to an angle of about 20 degrees. The patella is smelted with the new bone formed between the femur and tibia.

matism." That statement was correct then and it is right today. A metastatic joint infection will always lead to the destruction of the joint when it is initiated by a chill, and it means that virulent microorganisms are passing through the blood.

What happens then? After the initial chill this patient had a metastatic arthritis in both knees, one being more severe than the other. One leg was permitted to contract, the other was kept in a fairly straight position. The one that has been permitted to contract was the seat of the severe inflammatory condition, and now she has a complete bony ankylosis.

The factors of destruction of tissue in infections of the joints are similar to those causing destruction in other tissue infections, with the exception of the variations in vascularity in the synovial membrane and the comparative absence of vascularity in the fibrous capsule. They may be enumerated as follows:

1. Virulence of the microorganisms producing the infection.
2. The minus resistance of the patient.
3. Retention of infection products—pus under pressure, causing ischemia of the synovial surface, thus favoring the biotic and toxic necrotizing effects of the microorganisms.
4. The intra-articular pressure of the inflamed joint surfaces by the involuntary muscular contractions in its effort to immobilize the inflamed and painful joint.

The rôle played by each of these elements may vary in different joints and under different circumstances, but they essentially control all the destructions resulting from infections, whether (a) these occur as a result of lacerations and penetrations and direct infection admitted into the joint from without,—traumatic infections,—or whether (b) they are carried through the blood and lymph streams into the joint—hematogenous and lymphatic infections.

There are only two tissues in and at the joint that are the seats of primary infection, whether traumatic or hematogenous, and these are the synovial membrane and the bone. The cartilage and fibrous capsule are never the seats of primary infection.

In the traumatic cases, penetrations, punctures, etc., the infection is of the submesothelial layer after the mesothelial

cells have been destroyed by trauma or erosion. The hematogenous infections of the joint *never take place on the synovial surface*, but in the capillary and lymph layers of the synovial membrane, always beneath the endothelial layer.

In infection of the joint, either synovial or osseous, effusion takes place into the joint long before the microorganisms appear on the synovial surface or in the fluid, as in the following:

1. In tuberculosis of a bone in the neighborhood of a joint, effusion is present weeks or even months before the tubercle bacilli appear in the fluid of the joint cavity.

2. In metastatic osteomyelitis near a joint, effusion occurs and remains for weeks and months without the microorganisms ever appearing in the joint.

3. Periarticular infections of the pneumococcic type in children are accompanied by great articular effusion, but no pneumococci are present in the fluid.

4. In practically all metastatic infections of the joint the submesothelial layer is primarily involved, and holds the microorganisms for hours or days before they gain entrance to the fluid in the joint cavity. Streptococcic infection is an exception to this rule, as the cocci run freely through the lymph-spaces and enter the fluid of the joint early.

5. Cultures and smears made from fluid aspirated from inflamed joints giving negative results in the early stage, notwithstanding the great quantity of fluid present.

Aspiration of fluid in known Neisserian arthritides gives negative finding in from 20 to 93 per cent. of the cases, depending on the period of time that has elapsed between the appearance of the synovitis and the date of aspiration. This retention of the metastatic organism in the subendothelial layer accounts for the early negative bacterial findings in so many joint inflammations. In many of the arthritides we are, with our present knowledge, unable to locate the primary atrium of infection. They are termed cryptogenetic arthritides. In the majority of these cases the primary infection can be demonstrated clearly and unmistakably in some distant portion of the body.

It is my conviction that every type of non-traumatic joint in-

flammation is a metastatic manifestation of a primary infection in some other portion of the body. It is my further conviction that there is no idiopathic rheumatic arthritis, any more than there is an idiopathic peritonitis.

One of the reasons why the profession has been so tardy in recognizing the connection between the primary infections, as of the nose, pharynx, tonsils, skin, gall-bladder, typhoid ulcer, or urethra, and the secondary arthritides, is that a long period of time elapses between the primary infection and the secondary arthritides in a large percentage of the cases. In my analysis of hundreds of these cases I find that the Neisserian arthritis does not occur until eighteen to twenty-two days after the primary ocular, urethral, or vaginal infections, or a secondary exacerbation of the same. The influenzal infection of joints occurs between the tenth and the fourteenth days after the manifestation of the primary infection. Furuncular infections, usually of the staphylococcus type, show their metastases in the joints in the ninth to the eleventh days. Infections of the hand or foot of the mixed varieties, not streptococcic, usually show the joint metastasis about the tenth day, while the streptococcic infections can cause metastatic arthritides within forty-eight to seventy-two hours.

The scarlatinal arthritis infections usually occur from the fourth to the sixth day after the onset of symptoms. These are usually of streptococcic origin, and make their appearance about the time the original symptoms disappear. This period corresponds with many of the original infections of unidentified infective flora. The longest time is from the pharyngeal metastases of the disease.

In contrast with these we have the very late manifestation of the arthritic and osseous infections occurring in typhoid. These come on from the fourth to the eighth week, or about the time that the ulcers are healing or healed. The pneumonic joint metastases occur in about the middle of the time element, that is, from ten to sixteen days after onset, *i. e.*, in the period of pulmonary lysis. With the exception of the streptococcic group, the arthritides do not occur in the early stage of the primary infec-

tion, but always in the lytic or convalescent stage, or after the primary lesion has apparently healed.

Many of the joint localizations of the constitutional infection are associated with or are the results of—(1) trauma (most common of all); and (2) exposure, as wetting of the feet, playing in the snow, chilling of the general surface of the body, exposure and chilling in swimming, overexertion or long-continued exertion, and other factors so commonly recognized as focalizers of constitutional infections.

The term "rheumatism" should be expunged from medical nomenclature, and a name given to indicate in itself, at least, some of the pathologic or etiologic elements in the joints. I prefer the term "metastatic arthritis."

We are pleased to note that Dr. E. C. Rosenow is proving experimentally on animals what we have taught from clinical observation for eleven years. His work is very praiseworthy and instructive.

In the traumatic infections, Class A, the infective microorganisms are admitted to the surface of the synovial membrane by direct penetration. They do or do not produce a local infection early, dependent on whether the mesothelial cells are disturbed by the trauma or whether the infective material is held by the synovial membrane sufficiently long to erode the endothelial cells, which are shingles of protection and must be disturbed to admit the microorganisms into the submesothelial lymphatic and capillary zones.

In traumatic synovial lacerations unassociated with fractures the first indication for treatment is to close the joint capsules as accurately as possible without drainage. The subcutaneous cellular tissue down to the joint may be drained with a gutta-percha tissue. The joint, depending on its size, should immediately be injected with 10 to 20 c.c. of a 2 per cent. solution of liquor formaldehyd in glycerin. A Buck's extension should be applied, and the joint should be aspirated and reinjected in twenty-four hours if the tension is great, or if the temperature is ascending even without tension on the capsule. The injection or aspiration should be repeated in two, three, or four days, de-

pending on the constitutional manifestations of the presence or absence of infection.

Usually three or four injections suffice to sterilize the joint completely. In puncture wounds exactly the same details are followed, except that the aspiration, on account of recurrent tension in the joint, may need to be repeated more frequently, but the injection should not be more frequent.

The treatment should be instituted immediately after the injury if the best results are to be obtained. *No fluid and no treatment will restore joint structures that have already been destroyed by infection. Time is, therefore, an element of great importance.*

Hematogenous acute arthritides, from a surgical standpoint,—and they are all primarily surgical,—may be divided into—(a) monoarticular, biarticular, and triarticular types, with readily demonstrable primary foci of infection; (b) the polyarticular type, often of cryptogenetic origin. The first is usually initiated with a chill, followed by diffuse arthritic and muscular pains, and in twelve, twenty-four, or thirty-six hours there are severe pain and swelling in one or a few joints.

It is a clinical fact that this type often leads to a bony ankylosis of the joints involved, and the initial chill must be accepted as a warning that such a result is likely to follow. The microorganisms here are of the virulent pyogenic type, which destroy the synovial membrane and the cartilage, and finally lead to ankylosis—unless scientifically managed, from a surgical standpoint.

It is also a clinical fact, as I stated previously, that the polyarticular variety is rarely initiated with a chill, attacks many joints simultaneously, and rarely leads to bony ankylosis. It is the result of the milder types of microbic infection, and subsides in the course of six to eight weeks, leaving the joint in apparently as good condition as before. This result, however, is not uniform. Occasionally in the polyarticular variety, initiated with a chill, we have ankylosis of many of the larger joints, as the hip, knee, ankle, etc., and less frequently some of the small joints. Both of these varieties are, we believe, metastatic infections, and

in the great majority of cases we have been able to locate the primary atrium of infection.

In addition to these we have extensions into the joint from local bone diseases, as tuberculous osteitis and osteomyelitis, the former frequent and the most common cause of tuberculosis of the joint, the latter rare.

Infections of the joint from periarticular, communicating or contiguous phlegmons are uncommon. In one of my cases there was an infection of the subdeltoid bursa which subsequently involved the joint.

The suppurative and simple serous arthritides are the manifestations of the acute varieties of infections, as acute pharyngitis, paranasal sinus infections, paratonsillitis, acute gastrointestinal disorders, influenza, acute genito-urinary disorders, etc. The necrotizing, malignant, and ankylosing complications are the superlative degree of either of the preceding conditions in their advanced stages.

The fibroid, infiltrating, cicatrizing, and contracting variety are from the lower types of infection of the large intestine, gall-bladder, etc., and usually by the organisms of the colon group. The rheumatoid joints are most commonly metastatic from *chronic* infection of the alveolar processes, posterior nares, paranasal sinuses, tonsils, pharynx, genitalia, and sigmoid.

To revert to the case before us. The principle of an arthroplasty is just the same as that of a pseudo-arthritis. In fact it is, in a way, a pseudo-arthritis—an intentional one. We endeavor to produce, through surgery, in a clean-cut, definite way what nature does in a crude way in cases of non-union of fracture. That is all there is to it. Taking advantage of the knowledge we derived from observation and study of such pathologic conditions, led us to attempt the formation of new joints.

Our first operation of arthroplasty was performed in the early part of 1902. The patient, a boy, had a fractured hip from a bullet. I saw him last about nine years after the operation. He has a splendid result.

Our first case was, fortunately, a hip case. The hip cases are the easiest, next the elbow, next the shoulder, and last the

knee. The knee is the most difficult joint of all to subject to an arthroplasty. The operation is done on exact and definite mechanical lines. The technic of the operation on the different joints varies, not in principle, but only so far as is necessitated by the anatomy of the individual joint. The knee is, as I said, the most difficult joint in which to secure perfect restoration of function and of a nearly normal joint anatomy. Not only must the ankylosis be relieved, but the joint must be usable; the patient must be able to bear his weight on the leg and use it in locomotion without the assistance of cane or crutch. There must be no tendency to luxation in any direction, and the joint must be sufficiently firm to permit of its unsupported use. That means that the normal anatomic conformation of the joint must be restored as nearly as possible.

In this case today the joint had been transformed into a bony mass. The femur and the tibia are solidly united and the patella is fixed to the femur by a bony ankylosis. It is a superlative ankylosis.

In our original arthroplasties on the knee we made two lateral incisions, then we changed to a curved incision, convexity downward, encircling the patella. We had trouble in these cases with the skin-flap. We sometimes got a necrosis of the tip or margin of the flap, and now we have come back to the two lateral incisions. We make two interposing flaps, an internal and an external. Sometimes these flaps are of equal dimensions, and then, again, one, usually the internal, is larger than the other, depending on how much tissue, free from scar, there is that can be appropriated for this purpose. Originally I transplanted the fascia lata from over the vastus externus. Now I take the flaps from the inner and outer aspects of the knee, each flap including capsule, ligaments, and subcutaneous fat, except the two lateral ligaments which are posterior to the flap. The base of each flap is directed downward, and is left attached just below the base of each tibial tuberosity respectively. Our greatest trouble has been from the development of a hematoma in the tissue outside of the joint, due to the oozing from the bone. The question is whether we will not come to the use of drainage for forty-eight

to seventy-two hours in place of allowing the blood to accumulate and then aspirating it later. Drainage, I think, is the proper course to pursue. Whether we will do that in this case depends altogether on the conditions present at the close of the operation.

We will now proceed with the operation. We shall not place a constrictor on this leg because we wish to check all the bleeding as we progress. We expose the joint by means of two lateral incisions, one on either side of the patella, commencing one inch above and to the outer and inner upper angles of the patella, curving slightly inward and passing down to a point about four inches below the lower margin of the patella. These curvilinear incisions are from six to seven inches in length, separated a distance of four or five inches above and three inches below. The skin and superficial fascia is dissected up and the wound edges are retracted.

Now I am going to make my interposing flaps. You see the capsule has become a part of the periosteum. The external lateral ligaments have disappeared as separate and distinct ligaments. In making the interposing flaps, which measure about $2\frac{1}{2}$ inches in width and $3\frac{1}{2}$ inches in length, I dissect the tissues free down to the periosteum until I come to the posterior position where the external and internal ligaments should be, taking much of the fat and fascia and the capsule for my flap. This is mighty difficult. We always endeavor to get as much fat in these flaps as we can, and, fortunately, this patient is blessed with adipose tissue. We shall have to place a flap under the patella too. It will be dissected free from the fascia of the vastus externus.

The patella is adherent to the external condyloid ridge, which occurs more frequently than does union to the internal ridge. The external flap is much thicker in this case than the internal.

Now let us see if we can find the line of union between the patella and femur. By raising the skin-flap over the patella with a sharp hook I am able to see the line of union. The contraction of the quadriceps has forced this patella into the femur. See how nicely the patella shows as we elevate the skin-flap! We never divide the quadriceps tendon nor the ligamentum

patellæ. We free the patella from its union with the femur by means of a narrow straight carpenter's chisel. That gets our patella free.

We have handled this patella in a number of ways—by placing a flap under it to prevent its reunion with the femur, by splitting it into two parts from above downward and then turning the upper half under the lower so that the smooth aponeurotic surface came next to the femur, by rotating the patella or turning it turtle so that the prepatellar bursa rested on the femur, and by transplanting a detached flap of the trochanteric portion of the fascia lata between it and the femur. We have had good results with all these methods.

The next step is to divide the ankylosis between the tibia and femur and restore the conformation of the articular surface of both bones, at the same time removing enough bone to let the leg come around to a right angle with the thigh. We remove the bone from the upper end of the tibia. I will have to take off about three-fourths of an inch from the outer tuberosity and about five-eighths from the inner. I must preserve the intercondyloid ridge on the tibia, to maintain the stability of the joint and to prevent lateral luxation. You must leave that ridge, otherwise you will surely have a lateral displacement of the tibia. You must maintain the normal conformation of the joint as nearly as possible. I use a large curved carpenter's chisel in removing this bone.

Having located the line of union between the tibia and femur, we divide the ankylosis with the curved chisel, concavity directed upward, driving it in from the internal side of the joint first and then from the external. The assistant attempts to flex the joint as we do this, and now we have succeeded in freeing these bones. The curve of the chisel has restored the normal conformation of the articular surfaces of the condyles of the femur. The patella is not in the way because we retract it to one side or the other, as is needed, to secure free access to the joint. We do not touch the posterior portion of the capsule. The crucial ligaments are absent here because of the ankylosis. Remember the one structure to avoid laterally is the external popliteal nerve on the

outer side of the joint, and posteriorly the vessels and nerves in the popliteal spaces.

Now I will trim these bones to my liking with the chisels. The intercondyloid notch and ridge are properly matched and the bed for the patella is rounded out. On flexing the knee, we find that better than right angle flexion can be obtained, and on allowing the femur and tibia to come into position, we find that the leg is plumb. These raw bony surfaces must be smooth. Any little projecting spicule of bone would eventually interfere with the result.

All the posterior portion of the capsule remains. The crucial ligament attachment to each condyle is torn loose. That does not matter. What I depend on more than anything else is that plumb-line of the leg. It is going to be bowed forward a little because she had a slight curvature of the lower end of the femur, the result of the inflammatory condition which led to this ankylosis. The leg is going to be plumb as far as the lateral displacement is concerned. That is what I mean by "plumb."

The next question is the disposition of the patella. You see, there was a luxation outward of the patella, with the destruction of the normal patellar groove on the anterior surface of the femur. I have restored its normal bed by removing some of the bone with the curved chisel, and the patella goes back again to its place rather naturally. This patella will not be "turned turtle." I will interpose a flap of aponeurosis, fascia, and fat from the vastus externus, between the patella and the femur. I will in that way prevent the ankylosis from recurring. I will not need to shorten the quadriceps tendon or ligamentum patellæ.

We draw this fascial flap under the patella and suture it to the periosteum over the internal condyle of the femur and the fascia of the vastus internus. That covers all the raw bone surfaces here very effectively. We use chromicized catgut for suturing these flaps.

The next step of the operation is to place the interposing flaps between the tibia and the femur. I will place the external flap first. I anchor it with one stitch to the posterior capsule and the stump of the crucial ligament behind. A second stitch is

put through the tip of the flap. This stitch will later unite the two flaps in the middle line of the joint. A third stitch unites the anterior margin of the flap to the periosteum on the anterior edge of the tibia. The internal flap is similarly placed, and overlaps the external flap about an inch in the midline. The important factor is the fixing of the flaps. These flaps must cover all the raw bone surfaces, otherwise a bony union and a secondary ankylosis would result. The suture material is chromicized catgut.

The technic, you can see, is slow in execution; at the same time it is definite, clean-cut, and rather easily carried out. The two flaps overlap in the midline, and they form a splendid covering for the upper end of the tibia. The lower end of the femur does not need to be covered because it comes in contact with the flaps which cover the articular surface of the tibia.

This practically completes the operation, except for the closure. A few interrupted plain catgut sutures are put in on the inner and outer aspects of the joint, to approximate the deep tissues and give additional security to the joint. The skin incision will be closed with horsehair.

We stop all bleeding with a Blunk forceps. That is just purely a crushing instrument, and it does the work fairly well. In inflammatory tissues it cannot be used at all. In placing the deep sutures I will shorten the vastus internus. As you see, that pulls the patella over into normal position in the groove I made for it on the femur. Now we close the skin incision. You will note that there is considerable oozing from the wound; therefore we will put a rubber tissue drain in the lower angle of both wounds. That drain merely passes down to the joint, not into it. That oozing from the bone is a great source of annoyance. If we do not drain, a hematoma will form, and we must then aspirate later. This is the first case in which we have drained.

The doctor asks me if I do this operation in children. No, because you interfere with the epiphyseal line. If you remove the epiphysis of the bone, you stop the growth of the bone. We let the little patients go along to maturity before we operate for the bony ankylosis.

These drains will be taken out in seventy-two hours. I have tried to obliterate all dead space as thoroughly as I can, and get a contacting of this zone so as to get a primary agglutination and adhesion and a primary repair. That is done with the deep sutures. A Buck's extension with a 25-pound weight attached will be placed on this leg and left on for four weeks. An alcohol gauze dressing plus 1 per cent. of phenol will be applied, and the leg placed in a wire cage for support.

At the end of four weeks, possibly at the end of three weeks, depending on the condition of the soft parts, the patient starts lifting the knee—just lifting it by voluntary action. The skin stitches will be removed in about two weeks. We always use horsehair. We like it better than silkworm-gut. It is more elastic, and it does not cut the skin. We have used silkworm-gut and we have gone back again to horsehair. The patient will be up on crutches in about four weeks, and she will be encouraged to flex the knee as much as possible, but without putting any weight on the leg, for at least eight or ten weeks. We find that these cases do better when the patient moves the joint voluntarily, rather than to resort to forced flexion.

There is not much to show now that the work is done, but let me recapitulate the steps of the operation:

First: Make parallel incisions on the sides of the patella.

Second: Prepare the interposing flaps.

Third: When both of these flaps are prepared, free the patella by chiseling it from its point of attachment, either to the external or internal condyle.

Fourth: Free the attachment of the tibia to the femur.

Fifth: After that is freed and you have amply prepared the intercondyloid ridge on the tibia, then prepare the upper portion of both the external and internal tuberosity of the tibia and make two concavities that the condyles of the femur will fit into. When you get through see that all the posterior angle is free, otherwise you will leave a spiculum of bone sticking up on the intercondyloid elevation on the tibia.

Sixth: Then interpose the flaps, covering all the denuded bone.



Fig. 41B.—Complete bony ankylosis of right knee involving all the bones of the joint, with the leg flexed at an angle of 150 degrees.



Fig. 41C.—Skiagram made about one month after operation. The leg is straight and the joint cavity has been restored. The top of the tibia and fibula are on a line in the picture.



Fig. 41D.—Skiagram of both knees viewed from behind, the operated knee being on the right, the normal knee on the left. The joint cavity has been restored, together with the normal conformation of the bones. In the case of the operated knee the femur and tibia appear to be separated more widely than in the normal knee and the top of the fibula is nearer the articular surface of the tibia.



Fig. 41E.—Photograph made just before the operation, showing position of the leg.
The hip is not involved.



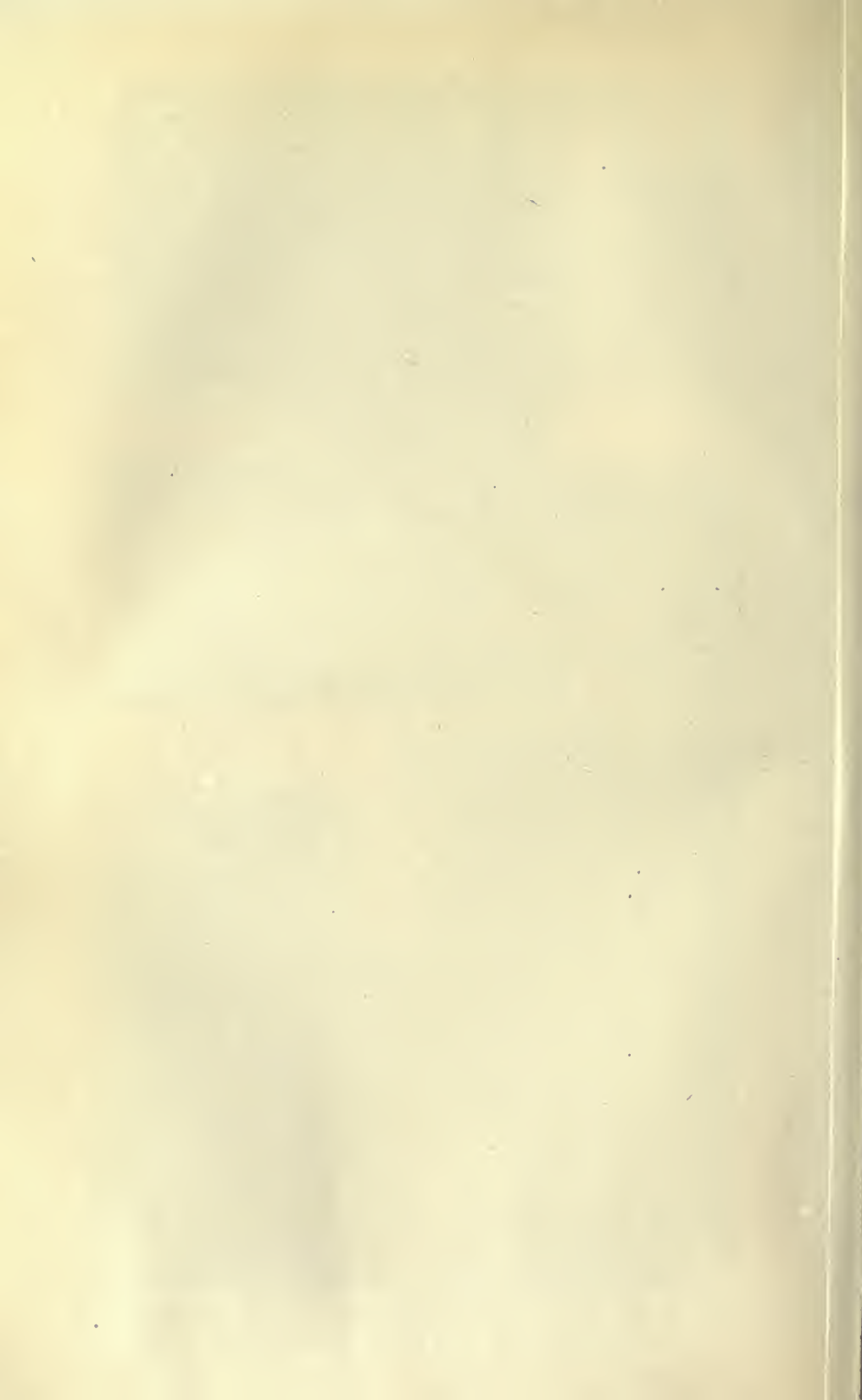
Figs. 41F, G, H.—Case VIII. Photographs made about five months after the operation. The patient had a normal range of motion in the joint and was able to walk without any assistance.



Fig. 41I.—Bony ankylosis of right knee involving femur and tibia only; the patella was free. The ankylosis extended the full width of the joint. The leg was straight.



Figs. 41J, 41K.—Skiagrams made four months after the operation, showing full restoration of the joint cavity and normal conformation of the femur and tibia.



Seventh: Place the subpatellar interposing flap.

Eighth: Close the wound by means of deep and superficial sutures.

We use a dressing moistened with alcohol and phenol to soak up the blood and to keep the material that escapes from the wounds sterile. I think it shortens the process of repair. An arthroplasty of the hip is done in one-third the time that it takes to do an arthroplasty of the knee.

Let me show you the result of one of our early cases. This young lady was operated on five years ago for a bony ankylosis of the right knee, with the knee flexed at an angle of about 150 degrees with the thigh. The ankylosis was the result of an insect-bite of the right hand and she had a metastatic infection in the right knee. The knee had been opened and finally was placed in a cast; then the adhesions were broken up a number of times, the final result being a complete bony ankylosis involving all the bones of the joint. (Figs. 41B-41H.)

We did a typical arthroplasty in this case, with the splitting of the patella, the external half being rotated and turned under the internal half. The patient was able to walk and sustain her full weight on the leg five weeks and six days after the operation. She could flex the limb almost to right angles eight weeks after the operation. She now has a full range of motion, and walks without any support and does not even limp. She has one-third of an inch elevation on the heel of her shoe to compensate for the shortening.

The skiagram shows that there is hardly any difference between the normal and the operated knee, and were it not for the high position of the head of the fibula of the knee operated on, one could easily make a mistake.

The interposing flap in this case consisted of the fascia lata with a thin layer of muscle removed from the outer surface of the vastus externus. This was one of our early cases, and we are very proud of the result.

I want to show you the pictures of another case in which there was a bony ankylosis between the tibia and the femur, the patella not being involved. The ankylosis extended the full

width of the joint, and the leg was straight. The ankylosis in that case was the result primarily of boils on the forearm. The infection extended to the upper end of the tibia by metastasis and to the knee by extension. She had many foci of infection, each one of which was incised and drained, including the knee-joint. (Figs. 41B-41K.)

When the patient first came to us there was a discharging sinus with a necrotic focus just below the patella. We refused to operate until this had healed. We removed the necrotic bone and got a complete healing. The patient came back to us nearly a year later. The leg was perfectly straight and stiff. The normal anatomy of the joint had been destroyed completely, but the patella was freely movable. She sustained the original infection a little over three years before we first saw her, and we performed the arthroplasty about four years after the primary infection from the boils and about two and one-half years after the knee first became involved. In this case we did not have to do anything with the patella. We simply placed two interposing flaps between the femur and the tibia.

The result in this case was a splendid one. The wound healed primarily, and the patient was up and around on crutches four weeks after the operation, with about 10 degrees of voluntary motion in the joint. When the patient left the hospital, about four months after the operation, she could flex her leg easily to right angles and was walking without any support whatever.

TUBERCULOSIS OF THE TESTICLE; ORCHIDECTOMY WITH IMPLANTATION OF PARAFFIN SUBSTITUTE FOR TESTIS

HISTORY

PATIENT, male, aged fifty-two, entered the hospital October 28, 1913, because of a swelling of the left testicle. The gland is adherent to the anterior surface of the scrotum. It is not tender or painful and is somewhat firm on palpation.

The present trouble dates back to June, 1911. While riding horseback the patient traumatized the left testicle. He immediately had severe pain in the testis, the pain continuing for a few minutes and then subsiding completely. He was perfectly well and healthy at the time of the injury, so far as he knew. During the following three weeks he did a lot of walking and climbing of stairs. At this time the right testicle was swollen and somewhat firm to touch, but not tender or painful. Occasionally, after much walking, he had a dragging sensation in the left testicle. Since that time the left testicle has fluctuated in size, becoming larger after much walking or exercise, and smaller after resting. It has never been painful or tender since the original injury. The patient has not had frequency of or painful urination. About two months ago he noticed that the left testicle was adherent to the scrotum in front. There has never been any abrasion of the skin of the scrotum. The right testicle is normal. Four days ago he consulted a physician who aspirated the testis and found pus.

Previous History.—He had malaria in 1882, without jaundice. The fever persisted for three weeks. He was shot through the right lung five years ago. Never had any night-sweats, but has a slight cough at present without much sputum, and it is free

from tubercle bacilli. He has not expectorated blood. Denies venereal infection.

Family History.—There is no history of tuberculosis or carcinoma in the family.

COMMENTS AND OPERATION

DR. MURPHY (October 30, 1913): What is the matter with him?

INTERN: He has some infection in his testicle.

DR. MURPHY: Yes.

INTERN: Possibly of the tubercular type.

DR. MURPHY: When did he have his last Neisserian infection?

INTERN: He never had a Neisserian infection.

DR. MURPHY: What are the varieties of metastatic infections which may occur in the testicle?

INTERN: He may have an infection from any part of the body.

DR. MURPHY: Yes. Mention them in the order of their frequency.

INTERN: First, the Neisserian infection; second, a tubercular infection; third, the metastases from other acute infectious diseases.

DR. MURPHY: Which infection is the most common?

INTERN: Pneumococcus.

DR. MURPHY: No, no, no! The most common metastatic infection of the testicle, excepting tuberculosis, is that following mumps. Next comes the infection associated with scarlet fever, measles, small-pox, typhus, influenza, typhoid, and pneumonia—in fact, any of the infectious diseases. Then comes the streptococcic type, which always occurs as a metastasis, and is usually associated with trauma of the testis. The infecting microorganisms may reach the testis by way of the blood-stream, as in the types just mentioned, or by way of the vas deferens from the urethra, prostate, and seminal vesicles. The most common of the invading organisms coming by this route are the gonococcus, staphylococcus, and streptococcus, and sometimes the tubercle bacillus reaches the testis by this route.

The next questions you ask yourself are: What tissue in the

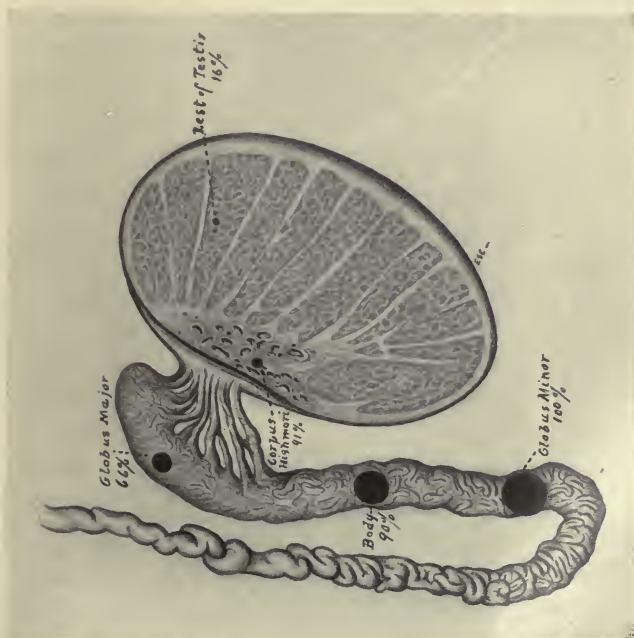


Fig. 42.—Tuberculosis of testicle. Diagrammatic representation of commonest sites of tuberculous deposits in the testicle, compiled from a series of tuberculous testicles removed by operation. The figures represent the percentage of cases which showed tuberculous deposits in that position. The size of the circles gives an approximation of the frequency with which tuberculous deposits are found in these sites (copied).

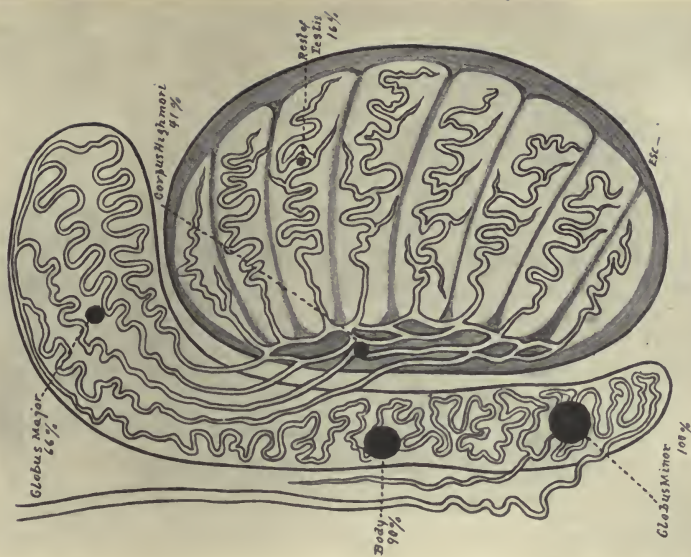


Fig. 43.—Tuberculosis of testicle. Schematic drawing of Fig. 42, showing testis and epididymis laid open (copied).

testis becomes involved primarily in these infections? In which tissue does the infection manifest itself—in the connective tissue or in the parenchyma? The most common form of acute epididymitis is the gonorrheal. It usually appears during the third, fourth, or fifth week of the gonorrhea, and is most often unilateral. Both testes are rarely involved at the same time. The gonococcus is carried from the urethra to the epididymis, along the vas deferens, by antiperistaltic contractions due to the irritation of the urethra. The testis proper is seldom affected. There may be an effusion into the tunica vaginalis. A Neisserian infection of the testicle is primarily an epididymis infection. I saw a young man yesterday who had had one testicle removed. He had an epididymitis following a gonorrhea, and the doctor removed the testicle. Never remove a testicle for a Neisserian infection of the epididymis. If a suppuration is present, the most you need to do is to remove the epididymis, leaving the testicle proper, because it has an internal as well as an external secretion.

Where does tuberculosis occur? It always occurs first in the epididymis, beginning either in the globus major or globus minor. If there is a mixed infection, if the patient has had gonorrhea, and we believe that is one of the predisposing causes to tuberculosis of the epididymis, an abscess may form and extend into the testicle. If it is an extensive suppuration, one which is permitted to go on for a long time, it may destroy all the secreting portion of the testicle within the tunica albuginea, but the hilum of the testicle is white fibrous tissue, and it does not admit the infection for a long period of time.

When you make a diagnosis of tuberculosis of the epididymis,—and the diagnosis can usually be made easily and early,—there is only one line of procedure, a removal of the epididymis, because it is going to be destroyed as a conducting tube, and there is the danger of the lesion destroying not only that testicle, but it may be disseminated into the other testicle. Furthermore, the continuous passage of the tuberculous material into the seminal vesicles often produces a tuberculosis of one or both seminal vesicles. Whether it progresses by way of the blood or by the route of the surface mucosa I do not know, but I believe it is a

hematogenous infection in a large percentage of the cases, because in many cases of tuberculosis of the epididymis there is no evidence of tuberculosis in any other part of the genito-urinary tract. Acute tuberculosis of the epididymis is an infrequent lesion.

In children the disease first affects the testis proper,—usually only one,—and it runs an acute course. The chronic form of tuberculosis of the epididymis and testis is the most common in adults, and may involve the other parts of the genito-urinary tract by an ascending infection. In the majority of cases it is secondary to pulmonary tuberculosis or to tuberculosis elsewhere in the body. As a rule, it is a unilateral affection, although both sides may be involved, either simultaneously or consecutively—in the latter case often after an interval of years. The disease usually begins in the form of scattered small tuberculous nodules. These increase in size, caseate, and coalesce until the whole epididymis is destroyed. Sooner or later the disease involves the testis proper by extension through necrosis.

As the disease progresses adhesions are formed with the tunica vaginalis, the scrotal tissues, and finally the skin. One or more discharging sinuses may form. If these sinuses are situated on the lower and back part of the scrotum, it is an evidence that the epididymis alone is involved. If they are situated on the anterior and upper parts of the scrotum, the testis proper is involved. The destructive process may become arrested at any stage, but, as a rule, it progresses until the testis is destroyed and other parts of the genito-urinary system may become involved, and a general tuberculosis result. Primary tuberculosis of the secreting portion of the testicle is always an evidence of a general miliary tuberculosis.

Orchitis as a complication of mumps usually occurs toward the end of the first week of the parotitis. The testis proper suddenly becomes painful and swollen. There is seldom any enlargement of the epididymis, but the scrotum becomes red and edematous. The condition is generally unilateral, although both testes may become involved consecutively, the inflammation in one having subsided before the other is affected. The condition

runs its course in four or five days. Suppuration rarely occurs, but atrophy of the testis is not uncommon. This is caused by the intertesticular pressure produced by the swollen and edematous seminiferous tubules inside the inelastic tunica albuginea, causing a rapid necrosis and later absorption.

The orchitis that occurs with the other acute infectious diseases resembles that associated with mumps, but is less acute. It usually occurs when the fever is at its height. Following scarlet fever there occur not infrequently a suppuration and an abscess in the testicle resulting in a complete destruction of the testicle. The local symptoms are apt to be overshadowed by those of the general disease, and the testicular lesion may be overlooked until the child has recovered from the scarlet fever. When you look for the testicle two or three months afterward, you may find only a button of tissue—just cicatricial tissue. The same type of infection occurs in association with small-pox, typhoid, typhus fever, and all that class of diseases.

The next class of infection of the testis is syphilis. This may take the form of a diffuse sclerosis of the intertubular connective tissue of the testis proper or of a gumma in the gland, or both conditions may be combined. When the sclerosis predominates, it tends to destroy the secreting tissue of the testis—the seminiferous tubules. The gummatous tissue tends to caseate and break down, leading to ulceration of the scrotum. Syphilis resembles sarcoma in the location of its primary focus. The rule is for sarcoma to take its origin in the connective tissue of the mediastinum testis, and syphilis occurs in practically the same tissue and in practically the same relation. That is why it is so difficult to make a differential diagnosis between syphilis and sarcoma of the testicle from the gross symptoms. With the discovery of the Wassermann test we hoped for great things in making a differential diagnosis between these two lesions, but the Wassermann test did not help us out nearly as much as we believed it would, because sarcoma may give a very marked reaction to that test, as does acute malaria. The luetin test is more reliable than the Wassermann in these cases.

Now we come to the consideration of tumors of the testicle.

Tumors of the testis are difficult to classify, because some of them are composed of different histologic elements, none of which predominates sufficiently to characterize the growth. Some tumors exhibit a marked tendency to undergo cystic and other forms of degeneration, hence lose their original characteristics. Another peculiarity of tumors of the testicle is their proneness to malignant transformation. Among the benign tumors of the testis are fibroma, myxoma, myoma, osteoma, and teratoma. Adenoma occurs practically only in combination with sarcomatous elements.

The malignant tumors of the testis are by far the most common. Two types of malignant disease occur in the testicle—one is carcinoma and the other is sarcoma. Carcinoma is rather uncommon, either as a primary or as a secondary lesion. The soft, medullary, or encephaloid carcinoma is the most frequent type of carcinoma of the testis. Sarcoma may occur primarily in the testicle or in the epididymis, but the testicle proper is the favorite situation. All varieties of sarcoma are met with. It is seldom possible to distinguish between a sarcoma and a carcinoma, either clinically or macroscopically.

Malignant disease of the testis may occur at any age. The majority of cases occur between twenty-five and forty-five. Sarcoma is not uncommon in children.

One of the most prominent characteristics of sarcoma of the testis is the rapidity and extent of the lymph-node infection.

When the left testis is involved, sarcoma travels through the blood-stream to its metastatic position in other parts of the body. When the right testis is involved, the metastases travel through the lymph-current. They lodge in the lymph-nodes of the groin, the postperitoneal lymphatics, and especially to the lymph-node on the renal vein.

The supraclavicular glands are sometimes infected by way of the lymph-stream. Metastasis sometimes occurs by way of the veins (most commonly on the right side), and secondary growths are found in the skin, lungs, and other viscera. Metastasis into the lymph-nodes on the left renal vein is very common in sarcoma, and unless one removes these lymph-nodes at the primary

operation, he is negligent of the best interests of the patient. When you find a tumor on the left renal vein that is palpable through the abdomen, do not attempt to take out the sarcoma, as it is already beyond the control of surgery. It is only in the early stage of involvement of these lymphatic nodes that the case is a favorable one for surgical intervention. The region of the kidney should always be examined in every case of sarcoma of the testis before the gland is removed.

Let us return to the case before us. There has been a supuration here. Why? Because the testis is fixed to the dartos and skin of the scrotum. We must make our incision wide of that zone and remove all the infective zone entirely. I will excise all the skin that might possibly be infected, so that we will insure a primary union. We will split this scrotal tissue down, keeping wide of the infection zone. When we first examined this scrotum we concluded that there had been a mixed infection. We inserted a hypodermic needle into this mass and withdrew some pus. Because this was a mixed infection we have decided on a removal of this testicle and an implantation of an artificial testicle—a paraffin substitute.

The field of operation has been prepared by scrubbing the skin with soap and water and a weak bichlorid solution. Now that the patient is asleep, we will cleanse the field thoroughly with alcohol. Never use alcohol or ether on the scrotum while the patient is awake. It causes too much pain. We make an elliptic incision, going wide of the infected zone, exposing the testis well and ligating all the bleeding points. We free the testis quite easily, and now we have the vas deferens and its vessels in view. The vas does not appear to be thickened enough for a tuberculosis. The para vas lymphatics are much infiltrated.

The next step is the care of the vas. We will separate the vas from the neighboring structures and tie these off separately. This vas is very hard and woody. We ligate the veins first and cut between two ligatures. Then we clamp, ligate, and divide the vas. We apply 95 per cent. phenol to the cut end of the vas for the purpose of cauterizing its mucosa. That lessens the risk

of infection with tubercle bacilli extending higher up, which is not at all uncommon.

Now the testis has been removed and we are getting ready to insert the substitute. We have to ligate every bleeding point. Why? Because we are putting in a foreign body—an artificial testicle. It is made of plain paraffin of a boiling-point of 122° F. I will place this testicle very high up, and I will secure it in place with a puckering string of catgut, including the edge of the dartos so as to make a sac or pocket for the foreign body so that it does not come near the infective zone at all. I have set it much higher up than the other testicle, but it will eventually slip down to a lower position in the scrotum. We will now suture the incision in the scrotum with horsehair, putting this stitch in as a mattress stitch. The wound will be dusted with bismuth subiodid and an ordinary plain sterile gauze dressing will be applied. The scrotum will be strapped up with adhesive plaster so that it will be supported properly.

Now let us examine this testicle and see whether there is a mixed infection in the epididymis, as well as a tuberculosis. Let us see whether it would have been possible for us to save this testicle. It would not because the infection has extended into the testis. This is infected tissue high up. I can feel some normal tissue here. In removing the epididymis you shell it off from the testicle, the same as you shell the adherent appendix from the caput coli.

On incising this gland I find that the tuberculosis has passed out of the epididymis into the mediastinum testis. It has penetrated through the tunica albuginea into the testicle proper. There is very little, if any, testicle proper left. It was a wise conclusion to decide on a removal of this testis, but we felt safe in doing so after we aspirated pus from it at the time of our original examination.

Let the record show that the lesion began in the epididymis, and that it extended by continuity through the tunica albuginea into the testis, and that an enormous abscess had formed within the testicle proper.

The abscess formed about a year and a half ago. Before that

time it would have been possible to remove that epididymis and leave the testis. If he gets manifestations in the other testicle, which is possible, then we will take out the epididymis at once and save the testicle. That is the advantage of seeing these cases of tuberculosis early and operating at once before the disease has extended into the testis. That is the advantage of knowing the real pathology in tuberculosis of the testicle. Remember that it is not a tuberculosis of the testicle proper: it is a tuberculosis of the epididymis.

The next question that comes up is, what will you do with the left seminal vesicle in this case if it is tuberculous. Nothing at all. Do not take it out. Just stop the source of supply of the tuberculous material and the seminal vesicle takes care of itself. It will cicatrize and eradicate the tuberculosis, or the focus will become encapsulated. The seminal vesicle heals as soon as you shut off the source of supply. If you remove it, the result will be a spread of the infection in the peritoneum or around the neck of the bladder.

The next proposition to consider is tuberculosis of the prostate. That is an entirely different problem. The prostate, as you know, is a muscular, fibrous, glandular structure, somewhat analogous to the uterus, except that the glandular elements in the prostate penetrate deeper and make up a larger amount of the organ than they do in the uterus. At the same time the glands have a very specific and definite function. When tuberculosis exists in any structure of that type, whether it is a tuberculosis of the uterus or a tuberculosis of the prostate, there is a feeble resistance on the part of these tissues against the tuberculous process. Why? Because the muscular tissue does not offer any resistance once it becomes involved in the tuberculous disease. The disease is never primarily a tuberculosis of the muscularis. It usually follows tuberculosis in the epididymis or seminal vesicles. When the prostate is involved by the tuberculosis, you must consider the proposition of removing not only the epididymis, but the prostate as well.

But there is another source of error, one that applies to lesions of the bladder as well as to tuberculous lesions of the prostate.

There may be a hemorrhage from the bladder in cases of tubercular epididymis, without tuberculous involvement of the kidney or bladder. This bleeding may continue for weeks and weeks and even months. You may be misled into regarding the cause of the bleeding as being a tuberculous ulcer of the bladder, but when you remove the epididymis, the hemorrhage ceases. The continued irritation from the epididymis is removed.

I recall the case of a clergyman on whom I operated twenty-five years ago. He had such a severe irritation of the bladder that he could not preach for more than fifteen or twenty minutes at a time without urinating. He was engaged in missionary work. He was practically totally incapacitated. We removed his epididymis and within three months every symptom of his bladder trouble had disappeared. Remember that was done a quarter of a century ago, and he has been well ever since, showing how perfect the repair was. He now weighs 280 pounds.

That gives us some idea as to the repair of tuberculosis of the bladder. Do not take out a piece of the bladder to make a differential diagnosis of the ulcer: just treat it on the basis that if it is a tuberculosis of the bladder it can heal without surgical removal of the ulcer. Tuberculosis heals from the assistance on the patient's part much more frequently than it does from the physician's aid. Performing any grave operation for tuberculosis in any tissue, as I have so frequently mentioned in reference to tuberculosis of the lung, lowers the patient's resistance and the reaction on the part of the tissues which is so necessary to getting a good result.

If the prostate is the seat of tuberculous disease, determine whether it is in the superlative or in a comparatively slight or moderate degree. If it is in the superlative degree, then an enucleation of the gland should be done, exactly as in the case of a tuberculous kidney. Remember in a tuberculosis of the kidney of the first degree a nephrectomy is not justified. In a tuberculosis of the kidney of the second and third degree nephrectomy should always be done. In the third degree it is absolutely indicated. Nephrectomy is contraindicated in bilateral renal tuberculosis, in non-tuberculous nephritis of the second kidney,

in tuberculous lesions of the bladder, unless the second kidney is healthy, tuberculous lesions of other organs, and when the condition of the patient is bad.

Let us consider now some of the essential features in connection with the etiology and symptomatology of this affection. The tubercle bacilli having gained entrance into the system, the next subject for consideration is: How do they find localization in the testes? There are a number of routes by which the infection may be carried to the sexual glands: (1) By the blood-stream; (2) along the surface of the mucous membrane of the genito-urinary tract; (3) by the lymphatics.

1. Transmission by the blood-stream has been, by most authors, considered to be the most important. The bacilli contained in the blood localize in the epididymis at some point where there exists a focus of diminished resistance, either congenital or produced by previous injury or disease. The frequent localization in the epididymis is accounted for by the fact that the spermatic artery divides opposite that organ, and that the vessels of the epididymis are smaller and more tortuous than those of the vas or testicle proper, the current therefore being slower.

2. In some cases the infection undoubtedly travels along the surface of the mucous membrane. Virchow has always held that the infection was a descending one, beginning in some of the higher genito-urinary organs. While we can undoubtedly have a descending infection, it is our observation, and that of many other investigators, that the disease is usually an ascending one, the epididymis being affected primarily, and the bladder, prostate, etc., secondarily, the process extending upward along the surface of the mucous membrane of the excretory duct.

3. Transmission of the infectious material to the testicle by the lymph-channels, while usually given as one of the routes, must be very rare, as the lymph current is directed away from the organ rather than toward it. No cases have been reported which show the infection to have taken place in this way. In from 73 to 75 per cent. of persons dying from all causes, mediastinal glandular tuberculosis is present, and this, in the majority of cases, is probably the atrium of infection in the cases of tes-

ticular disease. The latter is not usually associated with pulmonary tuberculosis, though Reclus observed that 50 per cent. presented lung findings, and 2.5 per cent. of the pulmonary cases over fifteen years of age suffered also from localization in the testicle. In his analysis of 500 cases of phthisis 64 had involvement of the genito-urinary tract, 45 of the genital tract, and in 19 the testicles alone were affected.

The symptoms vary materially in different patients, as in tuberculosis of other organs, varying with the rapidity of advancement of the process, the presence or absence of mixed infection, and again as the disease is primary or secondary in the testicle.

The symptoms of onset vary greatly at the different periods of life. In infants and young children the parents' attention is usually first called to the presence of a small nodule in the epididymis, the child having manifested no other symptoms, except perhaps slight tenderness in this region.

In adults the initial symptoms may be sudden or gradual in their onset, the latter being much the more common of the two. When associated with mixed infection, the onset is stormy, and there are all the manifestations of an acute epididymitis, resembling the gonorrheal form. Abscess formation may be rapid, and the pus find its way to the surface in a short time, leaving a sinus that heals slowly. Instead of breaking externally, the burrowing may be in the direction of the testis proper, and a portion or all of this organ be destroyed. Rupture into the cavity of the tunica vaginalis must be extremely rare, if it ever occurs.

When unassociated with mixed infection, the onset is slower, and many of the patients come to the physician for the first time because of the nodule which they have accidentally discovered in the epididymis. They are usually unable to say how long it has been there, but in some instances date its development from a slight injury or a gonorrheal epididymitis. The nodule or nodules are at first painless and only slightly tender on compression; but usually after a short time the patient experiences a dull, aching pain in the testicle, brought on or aggravated by exercise.

This pain may be referred to the testicle or upward along the spermatic cord, sometimes even extending into the iliolumbar region of the same side. It is rarely severe, and may be entirely absent when the patient is at rest.

A urethral discharge, usually whitish and mucoid in character, has been noted in a considerable proportion of the cases, usually quite early in the course of the disease. Instead of being white, it may be purulent or even bloody. Various theories have been advanced as to the origin of this discharge, some claiming that it is due to tuberculosis of the prostate, vesiculæ seminales, or the prostatic urethra, while others believe it is caused by a catarrhal condition of the posterior urethra and seminal vesicles. The latter is probably the true explanation, as it disappears after castration or epididymectomy. Symptoms of vesical irritation are present in the great majority of cases without mixed infection, and they may be the first to attract the patient's attention. At first there may be slightly increased frequency of urination, with perhaps a little burning during the act, referred to the base of the bladder. As the case progresses the irritability becomes more marked until the patient is unable to retain his urine more than fifteen or twenty minutes at a time, involuntary passage taking place if he attempts to do so. Later, also, tenesmus develops and may become very severe. The fact that vesical symptoms are usually absent in the cases with secondary infection is to be explained, in all probability, by the edema and swelling of the vas, which does not permit the irritating material to pass through it to the vesiculæ seminales.

Hematuria may occur early or late, and is usually not profuse. The blood is passed with the urine, and without obvious cause, in this latter respect differing from the hemorrhage of renal calculus, which is brought on by severe exercise, such as horse-back riding, etc. The source and cause of this hemorrhage are somewhat uncertain, but we believe it is due, as are the symptoms of vesical irritation, to the irritating action of the products of the tubercular process, which are discharged into the prostatic urethra through the vesiculæ seminales. This discharge produces a catarrhal condition of the mucous membrane in the

posterior urethra and trigone of the bladder, with, in many cases, erosions and superficial ulcerations. That the vesical irritation and hemorrhage are not manifestations of a tubercular process in the bladder and prostate we are convinced from clinical observation, as in almost all cases in which these symptoms are present there is an immediate cessation of them after castration or resection of the epididymis, which could not possibly be the case if they were due to tuberculosis of these parts.

Hydrocele, which is present in about one-third of the cases, may have been the first symptom to attract the patient's attention. It is rarely large, when due to tuberculosis in the epididymis, and there is but little difficulty presented in diagnosis.

At varying intervals from the onset of the trouble softening of the deposit in the epididymis takes place. This, in the acute cases, may be a few weeks, while in the more chronic ones it may not occur for months or even years after the onset. When the abscess is about to rupture, the skin of the scrotum over the fluctuating mass becomes adherent to it, bluish in color, and finally perforates by a small opening, through which the characteristic caseous material is discharged. There is usually no pain attending this process and but little tenderness, except where it is associated with mixed infection, in which event we have all the manifestations of an acute cellulitis. The sinuses formed by rupture of the abscess cavity may be single or multiple, and situated in different parts of the scrotum. They usually remain open indefinitely, and show little tendency to close spontaneously. The amount of discharge is slight.

Hernia testis is rarely seen at the present time, probably because the cases are operated on at an early stage. It is consequent upon the rupture of a tubercular abscess situated in the testicle proper, and consists simply of tuberculous granulation tissue, which is extruded through the opening, forming a fungus mass of greater or less size.

Constitutional symptoms are present in the majority of cases, though they differ greatly in degree. Some patients present the typical tubercular appearance, while others appear to be in perfect general health. In most of the cases, after the process has been

present for some time, there is loss of weight and strength, with perhaps some evening temperature, rarely exceeding 100° F., and night-sweats.

The sexual desire is usually unaffected, except in the very severe cases, even though, when both epididymi are involved, there may be no seminal discharge during intercourse.

Examination of the patient may reveal the presence of some old tubercular lesions on other parts of the body, such as lupus scars, cicatrices of the neck, or perhaps the evidences of a healed tuberculous osteitis.

Rectal examination, for the purpose of ascertaining the condition of the vesiculæ seminales and prostate, should always be made. The seminal vesicles are frequently involved, or if only one is affected, it is more likely to be the one on the side of the affected epididymis. In the early stages the vesicle is soft, swollen, somewhat painful to pressure, and can be outlined throughout its entire length by the examining finger. Later, nodules develop in it, and it presents a very irregular shape, hard, with, perhaps, fluctuating areas in different parts. When both are affected, they can be plainly felt converging toward the prostatic urethra.

It is more difficult to determine tubercular disease of the prostate, especially in its early stages, because the process is usually situated deep in the substance of the gland. In the early stage of the prostatic disease the gland may be somewhat swollen and tender to pressure, while later, when the process is farther advanced, it may be nodular and present fluctuating areas. Either lateral lobe may be affected singly, or if the entire gland is involved, it may be more advanced on one side than on the other. The inguinal lymph-nodes are rarely involved in tubercular disease of the testicle.

The condition of the urine will depend on the stage to which the disease has advanced. Usually when the patient presents himself for treatment small quantities of pus and blood will be found in the centrifuged specimen. By staining the pus for tubercle bacilli they can usually be demonstrated, although almost always in very small numbers.

The routes by which extension of the tubercular process takes place are at present the subject of much dispute, many authors claiming that the disease exists primarily above and extends downward to the epididymis along the surface of the mucous membrane, extension, therefore, in an upward direction not taking place. It is our opinion that the primary focus is in the epididymis, and that subsequently extension takes place along the lumen of the vas to the vesiculæ seminales, prostate, and in a few cases to the bladder, ureters, and kidney. Pott's disease of the spine we have frequently observed following tuberculosis of the testicle, and this, as well as the cases of general miliary tuberculosis which occasionally develop, must be explained by dissemination of the infectious material through the blood-stream.

As to the differential diagnosis, in acute cases, where the symptoms come on suddenly and with great intensity, the diagnosis from gonorrheal epididymitis may, at first, be difficult. The principal points to be considered are:

The presence of an active gonorrhea, the discharge of which may have suddenly disappeared just before the onset of swelling in the epididymis.

Examination of opposite epididymis, vesiculæ seminales, and prostate shows absence of nodules.

Previous history of patient: In tubercular disease there is frequently a history of recurring mild attacks of epididymitis, or enlarged cervical glands in childhood, osteomyelitis, etc. These are more likely to be absent in the gonorrheal cases.

Occasionally it may be impossible to make an immediate diagnosis, and in these we must await further developments, which will occur after the acute swelling has subsided. The tuberculin test might be valuable here, and in all cases where a urethral discharge is present repeated bacteriologic examinations should be made and guinea-pig inoculation tests instituted.

The table which we have had placed on the board presents the essential points in the differential diagnosis between some of these lesions:

TUMORS OF THE TESTICLE

	CHRONIC EPIDIDYMITIS	TUBERCULOSIS	SYPHILIS	TUMOR
History.....	Gonorrhea, stricture, hypertrophy of prostate.	Tuberculosis, family or personal.	Syphilis, inherited or acquired.	Perhaps trauma.
Frequency.....	Uncommon.	Frequent.	Frequent.	Rare.
Size.....	Small between attacks.	Does not reach any great size.	Does not reach any great size.	May reach any size.
Tenderness.....	Yes.	Yes.	No.	No.
Shape.....	Between attacks testis is normal; epididymis nodular.	Epididymis nodular. Testis not involved except in acute or old chronic cases.	Testis evenly enlarged; slightly nodular epididymis.	Testis greatly enlarged; no characteristic involvement of epididymis.
Cord.....	May be slightly thickened.	Enlarged and nodular.	Free.	Free.
Seminal vesicles.	Usually distended.	Tuberculous.	Uninfluenced.	Uninfluenced.
Prostate.....	Posterior urethra inflamed.	Congested or tuberculous.	Uninfluenced.	Uninfluenced.
Urine.....	Cloudy.	Cloudy, may contain bacilli.	Clear.	Clear.
Hydrocele.....	Unusual.	Often.	Nearly always.	Unusual.
Onset.....	Usually acute.	Usually chronic.	Chronic.	Chronic.
Age.....	Adult life.	Not often after thirty.	Middle life.	Any age.
Origin.....	Epididymis.	Epididymis.	Testis.	Testis.
Course.....	Recurring acute attacks.	Chronic.	Very chronic.	Usually rapid.
Suppuration....	Unusual.	Common.	Rare.	None.
Atrophy of testis	Rare.	Rare.	Common.	Never.
Opposite testicle	Often involved.	Usually involved subsequently.	Free.	Free.

In tuberculosis of the testicle the prognosis depends upon: (1) The age of the patient; (2) whether the infection is simple or of a mixed character; (3) the location of the primary focus of infection; (4) whether or not other portions of the genito-urinary tract or more distant parts of the body are involved.

The prognosis in children in localized disease of the epididymis, or even where it has extended into the testicle proper, with or without mixed infection, is usually favorable. Encapsulation takes place in the majority of cases in absence of pus infection, and a portion of the testis is preserved. Even where secondary infection has taken place and the abscess cavity discharged externally, we can always hope for the preservation of at least a small portion of the glandular structure. This tendency in children to encapsulation and limitation of the process is the same as that observed in tuberculosis of other organs in them, with the exceptions of tubercular meningitis and, to a lesser degree, tuberculous osteitis.

In adult cases many complications are apt to develop, and it

is on these that the prognosis will in large part depend. The most important are: (1) Abscess formation with resulting sinuses of the scrotum; (2) involvement of the seminal vesicles and prostate; (3) tuberculosis of the bladder (rare). In addition to these we may have foci develop in any other part of the body, and even general miliary tuberculosis has been known to follow a primary lesion in the epididymis. Whether the affection of the seminal vesicles or prostate precedes or follows the testicular disease or not, the clinical fact remains the same. The removal of the testicle or epididymis causes in a large percentage of the cases a complete subsidence of the vesical and prostatic symptoms and a healing of the tubercular process in these parts. From the symptoms presented by some of the patients who have come under our care it seemed certain that the tuberculosis had extended to the mucosa of the bladder, but on removal of the diseased testicle or epididymis they entirely subsided. While the prognosis is favorable in prostatic and seminal vesicle involvement, it is distinctly unfavorable when the disease has extended to the bladder. In these cases it very frequently further extends to the ureters and kidneys, and the patient soon succumbs to renal or general miliary tuberculosis.

If the epididymis, which is primarily affected, be removed early, it is probable that the other testicle will not become involved. Should it, however, become implicated, the urinary and vesical symptoms will recur and bacilli will again be found in the urine.

[The subsequent course of this case was uneventful. The wound healed by first intention.—ED.]

CHARCOT ANKLE; REMOVAL OF ARTICULATION AND NAILING OF ASTRAGALUS TO TIBIA

HISTORY

PATIENT, male, aged forty, entered the hospital November 12, 1913, because of deformity of the right ankle and right metacarpophalangeal joint of large toe, which interfered with his walking.

Present trouble dates back to two years ago, when the patient made a misstep which caused him to fall, and while falling he heard a clicking noise in the right ankle. This was followed by severe pain in the ankle. He got up without assistance, but the ankle was weak, and he had severe pain in it when he attempted to put weight on the leg. The next morning, on awakening, the ankle-joint was greatly swollen and tender, and he remained in bed. A doctor was called the next day, and he pronounced it a fracture and advised cupping, which was done. A liniment was also applied. One week later a plaster cast was applied, extending from the base of the toes to just below the knee. This was left on for eight or ten weeks, the patient remaining in bed during this time. When the cast was removed, a bilateral splint was applied. This was left on for two weeks.

When the splint was removed, he got out of bed and tried to walk, but he could not put any weight on the leg, both because of the weakness and the pain in the ankle-joint. He also noticed a great deformity of the ankle. He used crutches for a short time, then discarded them, and continued to use the leg. Since then the ankle has been becoming more and more deformed, but less painful. At present there is no pain, but a marked deformity. No other joints were involved. A Wassermann test was made and found positive. He also has an absence of the patellar reflexes, an Argyll-Robertson pupil, and the Romberg sign.

Previous History.—He had typhoid at twenty-two; no complications; rheumatism seven years ago. Has one child living and well, aged nine years. Wife has had no miscarriages. His left leg was amputated below the knee two years ago because of a compound fracture which failed to heal. He denies venereal infection and lues.

Family History.—Negative as to tuberculosis and carcinoma.

COMMENTS AND OPERATION

DR. MURPHY: What is the matter with him?

INTERN: He has a Charcot ankle.

DR. MURPHY: What is there in that history to make you think it is a Charcot joint?

INTERN: He had a positive Wassermann.

VISITING DOCTOR: The absence of pain at this time notwithstanding the deformity.

DR. MURPHY: That is just what the intern said he did not have. The history is wrong. The doctor here is right. He did not have pain in his ankle even after it fractured. That is one of the peculiarities of a Charcot joint—the patient does not have pain where he sustains a fracture, and this man told me that he had no pain. The intern thought he should have pain to fit the injury, while the real fact is that he had no severe pain.

Let me tell you the story of three of these cases. A man came to me severely criticizing his doctor. He said that he had had a Pott's fracture, but on examination we made a diagnosis of a Charcot ankle with great lateral displacement. The foot was inverted, owing to the absorption of a large portion of the lower end of the tibia and an erosion of the astragalus. A Pott's fracture is always an eversion fracture; the displacement of the foot is outward, not inward, as in this case. The man whose case I am citing fell a distance of about 20 feet, landed on his right leg, and fractured his ankle. The fracture was not compound. He did not have any pain then or afterward. His doctor immediately put on a circular plaster cast, including the foot and leg to the knee. It was removed after seven weeks, and the patient permitted to walk on crutches without touching the in-

jured foot to the ground. The ankle appeared to be in good position, and he could move his foot without causing pain at the time of removing the cast.

Five weeks after the removal of the cast he was permitted to put some weight on the leg, and at the end of another five weeks he discarded the crutches and went to work. His foot was still in good position. He was on his feet nearly all day. Within two weeks he had a little pain in the ankle on walking, then the ankle began to swell. Soon the leg became deformed gradually. The external malleolus became very prominent and came closer to the ground. The foot was luxated inward, although it was flat on the ground. It seemed to be crowded up into the tibia.

The swelling and deformity increased in severity until finally he could not walk on the leg. He went to bed for a few weeks and the pain and swelling disappeared, but the deformity remained.

That told the story. I knew that it was something else than a Pott's fracture. I asked him how much pain he had when he broke the ankle. He said, "Why doctor I never had any pain." That told the story. He had a positive Wassermann. It was a Charcot ankle. (See Figs. 7-14, pp. 15-18.)

We had another similar case—a Charcot hip. The skiagram showed an enormous deposit of bone extending from the upper margin of the femur to the anterior superior spine of the ilium. His doctor had made a diagnosis of sarcoma, because this mass of bone looked like a sarcoma, and I nearly fell into the error myself. As the patient started to leave my office I noticed that he limped. I said, "Why do you limp? Have you pain in your hip?" He said, "No, I never had any pain in the hip." That put me on the right track. He was riding a bicycle, and fell off, striking on his left hip. He had no pain. He got back on his bicycle and rode home. He even worked for two weeks afterward. Although he limped, he was free from pain. The only complaint he made was that he could not control this leg as well as formerly. He never had pain, even on motion. That told the story—that the fracture was a pathologic fracture, the result of a bone lesion, which in his case was syphilitic in origin.

He had the Argyll-Robertson pupil, a positive Wassermann, and all the other symptoms of syphilis. The skiagram showed a fracture of the neck of the femur, with an enormous ossification of the fibrous layer of the capsule. (See Figs. 18-20, pp. 22 and 23.)

The doctor, in reading the history of the case before us, said the patient denied syphilis, but he had a Romberg, an Argyll-Robertson pupil, the fixed pupils on both sides, the absence of the tendon-reflexes, and a positive Wassermann. The patient said he did not have pain. That tells the story. You must be careful of the Wassermann test, because not infrequently you get a positive Wassermann in sarcoma without syphilis being present. We have had half a dozen cases of that kind, some of them with a triple plus reaction, which leads us to ask some serious questions regarding the value of the Wassermann.

Now, then, what are we going to do in the management of this case? That foot is out of commission. What can you do? You can expose the ankle, take off the lower articular surface of the tibia and the upper articular surface of the astragalus, freshen the surfaces of these bones, place them in accurate apposition, and then fix them by driving a 14- or 16-penny nail into the sole of the foot through the os calcis and astragalus into the tibia, and you get a bony ankylosis. He will have a stiff ankle, but a leg that he can walk on without any hazard. A bony union takes place very rapidly in these cases.

This man is out of commission. He has lost part of the other leg, and you see how important it is to give him at least one good firm leg to balance himself with.

VISITING DOCTOR: Is this patient given any other treatment than this operation?

DR. MURPHY: Yes. He will be given neosalvarsan.

VISITING DOCTOR: Could this present condition have been avoided by treatment when it first began?

DR. MURPHY: That is rather a difficult question to answer. I think not. Treatment for syphilis would probably have been beneficial, but once these Charcot joints start they are almost beyond treatment other than surgical. I have not seen a case

where I could stay the pathologic process. Extension would not help except, probably, to prevent the severe deformity.

Arthrodesis is the most desirable operation than can be performed in these cases. It gives a firm fixed bony support, stops the local irritation, and cures the disease. The reason it is not more popular is that many have failed to get a bony ankylosis. I believe that this failure must be charged to a deficient removal of the capsule and cartilage of the joint, defective fixation of the bone, insufficient or absence of bony contacting. The ends of the bones must be well freshened, contacted, and fixed. Mere cureting of the diseased joint is not sufficient. All the cartilage and bone must be removed with the chisel. We will expose the joint by a longitudinal incision over the internal malleolus posterior to the tendon of the tibialis anticus. Retracting the wound edges well, you can see the extent of the destruction of this joint.

Note how greatly thickened the periosteum is. We must be careful in this dissection because we are coming into the neighborhood of the posterior tibial artery and nerve. Now the tibio-astragaloid joint is opened. See the fluid coming out! There is always an excess of fluid in these cases. That is all cancellated bone, you see, the result of trophic changes. See the cartilaginous formation there at the margin! That is typical of these cases. This bone is very greatly enlarged. When we get through here the leg will be short, but that will not matter with him, if we can get him to walk with his foot flat. The other leg, which is an artificial one, can be made to any length.

What we want to do here is to give this man a foot which will be of some service to him—a foot which he can use with some comfort. We are going to remove the internal and external malleolus and then as much of the tibia as is necessary to get to normal bone. The same will be done with the upper surface of the astragalus and the lower end of the fibula. We are now removing the end of the tibia with the chisel. I find that an inch and a half had to be removed. The bone is normal here. The interosseous ligament is intact, and we must keep it so to maintain the apposition of the tibia and fibula. We will now

remove half an inch of the upper surface of the astragalus. That gives us two good surfaces for contact and the production of an ankylosis between the tibia and the astragalus. The foot is now at a right angle to the tibia and just slightly inverted. This position will be maintained by means of two 16-penny nails driven into the sole of the foot, through the os calcis, and astragalus into the tibia. The skin is first nicked with the tenotome and then the nails are driven home—one about one inch behind and slightly internal to the other, so that there will be no chance for a rotation or sliding of the tibia.

VISITING DOCTOR: Why would not a fracture unite as well as an arthrodesis would?

DR. MURPHY: In the beginning, because of the close proximity of the fracture to the joint, immobilization was not possible. If you can keep the fragments completely immobilized, it would be all right, but when the fracture is near a joint, that is not possible. Fractures in the shafts of bones in these tabetic cases unite quite readily if they are properly immobilized.

Let the record show that the external and internal malleoli were removed with the chisel; that the ends of the bone were freshened; that after putting the foot in the proper position two nails were driven through the sole of the foot up into the tibia.

We close the wound with horsehair, put on the usual dressing and a plaster-of-Paris cast extending from the toes to the knee.

[The case progressed favorably.—Ed.]

LORD LISTER AND ANTISEPTIC SURGERY

A Talk by Sir Rickman J. Godlee, President of the
Royal College of Surgeons of England, at the
Clinic held on Monday, November 10, 1913

DR. MURPHY: We are honored in having with us today a man who occupies the highest official position in connection with scientific medicine of any man in the world; a man who has been not only in close touch with, but a part of, the evolution of antiseptic surgery and a part of aseptic surgery up to the present time; a man who, on account of his personal relations with the late Lord Lister, can tell us more of the trials and hardships, as well as the rewards and successes, of the greatest man of surgery of all time than any other man; a man who has personally contributed to so many lines of successful surgery: I have the pleasure of presenting to you Sir Rickman J. Godlee, President of the Royal College of Surgeons of England, who will now talk to you.

SIR GODLEE: Gentlemen: I thank you very much for that hearty cheer and hearty welcome from this large party of American surgeons to a really insignificant stranger from afar. I wish to say, after my experience this morning, and the words that have been addressed to you in regard to myself, a few words about Dr. Murphy himself. Dr. Murphy is a celebrated personage. He is also, if I may be allowed the expression, a notorious character. I will tell you why he is a celebrated personage. It is because he is one of the most original, one of the most ingenious, one of the most eloquent, and one of the most distinguished surgeons of the present day. I have had an opportunity of seeing this illustrated by the way in which he has presented his cases to us this morning, and I had already heard of his reputation before leaving my own country. It was said to me the other day, by an American surgeon, in speaking about Dr. Murphy, that every one who came

in contact with him came under his spell. He said that Dr. Murphy will describe a case of Colles' fracture in such a way that you might think that he had discovered the displacement and the deformities himself, and will crane your necks forward to hear him as if he were describing a new discovery. I will tell you why he is a notorious character. He is notorious because he is in the habit of enticing simple people like myself into his clinic and forcing them to speak to his class on any subject which he intrusts to them. Some one said to me the other day: "If Murphy asks you to do a thing, you had better do it at once, because he will force you to do it in the end."

Gentlemen, the temptation is to say something silly, but this, I know, would not do because I heard on the other side of the Atlantic that there would be a stenographer taking down every word that is said. He has asked me to speak as one of those who was acquainted with surgical work as it existed before Lister's discoveries. This, fortunately, I am able to do, because although Lister had published his first papers on Antiseptic Surgery before I was a student at University College in the early seventies, the practice had not been modified to any extent. Indeed, it was long before his work was accepted in London and other parts of England, although it was enthusiastically received on the continent of Europe. I am not able to speak with any certainty as to the time when antiseptic surgery was adopted in America. I will, therefore, briefly describe the condition of things which existed in the wards and in the theater of University College Hospital at the time I am speaking about. They were very much such as would have been found twenty years before, when Lister was a student at the same hospital. The walls of the dingy wards were covered with pictures which accumulated dust. The washing arrangements consisted of a basin and a jug at each end of the table, and there were a few unsterilized towels. A single probe circulated from patient to patient, washed simply in unboiled water. Many of the patients were dressed with linseed poultices, and most of them suppurated, and, indeed, none of them healed by first intention except wounds that were made on the face. I think of a case of compound fracture of both bones

of the leg which we endeavored to treat according to the antiseptic methods employed at the time. The leg was washed with a solution of carbolic acid 1 : 20, and the wound was forcibly injected with the same material. The wound was covered with a piece of lac plaster. We had failed to examine carefully the back of the limb, where the slough caused by the original injury took place, with the result that the whole wound suppurated and the limb had to be amputated. I recollect my chief, Mr. Erichsen, saying to me after this: "Now, Godlee, no more antiseptics here." As in this case, compound fractures were almost always followed by amputation. Even compound fractures with small wounds suppurated.

It is interesting, after what Dr. Murphy said this morning about his treatment of compound fractures, that although the results in those days were so disastrous, our method of treating them was apparently almost exactly the same except that the materials used for washing the limb had not been previously sterilized. It was this fatality in connection with compound fractures that led Lister to say, when contemplating a case of comminuted fracture with unbroken skin, that if any one could discover what it was that made the difference between the behavior of such a case when the skin was broken and when it was intact, he would be a benefactor to his race. The operating theater was a much smaller one than this; the washing arrangement consisted of a single basin; our sister who attended to the sponges was not dressed like yours, in a sterilized gown, but in black flannel; the instruments were not boiled; the surgeon used no antiseptics, and the skin of the patient was not purified in any way. Whip-cord was used for tying the vessels, the ends of which were allowed to come out of the corner of the wound as a drain. These ligatures remained for several days, were pulled out by the dresser when they became loose, with the exception of one, to which a knot had been applied, indicating that it was attached to the main artery, which was reserved for the special attention of the house surgeon. You could easily imagine that the mortality from operations done in this manner was very large, and that the number of operations done was insignificant as com-

pared with those performed at the present day. Hernias were never operated upon unless they were strangulated, and many of them died from peritonitis. Practically all wounds suppurated, so that a period of fever followed the operation.

Well, gentlemen, Dr. Murphy has asked me to say something about the early struggles of Lord Lister in bringing his discovery before the world, but this is really too well known, and in spite of his injunctions, I will not attempt to go over this ground again, but will pass on to say something about the methods which Lister used at the end of his surgical career. I was fortunate enough to assist him in his private practice for many years, and I have often seen him operate at King's College Hospital.

In this hospital the theater was large and dark, and had none of the modern refinements which adorn our operative theaters today, but I need not say that excellent work was done in them. His methods have been followed by Sir Watson Cheyne in the same theater up until a few weeks ago with equal success. Lister did not boil his instruments: they were simply placed in a solution of carbolic acid 1:20. He wore no gloves, and his hands, which it should be added were always rough, were sterilized by a mixture of 1:20 carbolic acid in 1:500 sublimate solution. This goes by the name of the "strong mixture" at King's College Hospital, and it is much too strong for ordinary hands. Lister used marine sponges which were purified in carbolic acid solution. In his early days his sponges after an operation were placed in a tank of water, where the small amount of remaining blood contained in them was decomposed. Before the operation these sponges were taken out and washed in carbolic acid lotion, and he used to hold that this purification was sufficient.

Coming back to King's College Hospital, I want to impress upon you that Lister trusted practically altogether chemical antiseptics. He was, of course, well aware of the injury done to the human tissues by the use of these materials, and he reduced the strength of them to the minimum which was consistent with safety. Indeed, he was the first to try to apply what is now known as aseptic surgery, but he concluded that it was less safe and less easy for those who are not working under the favorable

circumstances which exist in modern hospitals of the present day. He maintained that the disadvantage of a dressing which contains no antiseptic material is that it fails to prevent the infection which may arise after an operation from the skin; then if it becomes soaked through with discharges, the infection may spread from the outside through the dressing to the wound. He, therefore, always employed a dressing containing some trustworthy antiseptic material.

What have been my reasons for comparing Lister's practice with that of those who work amid all the polished refinements of the modern hospital? It is in order to emphasize the fact that while the latter are really trusting to that most powerful of all antiseptics,—heat,—any one who is working under less favorable circumstances, say, in the cottages of the poor, can obtain equally good results by making use of chemical antiseptics. That, I feel, is the sort of message which Lord Lister would wish me to convey in speaking to American surgeons, many of whom are practising in the remote parts, far away from the surgical luxuries which we meet with in all the great hospitals of this city.

NITROUS OXID ANESTHESIA

A Talk by Dr. George W. Crile, of Cleveland, Ohio,
at the Clinic held on Friday, November 14, 1913

DR. MURPHY: While I am doing that work, we have with us also a man who is known wherever surgery is known, not alone for his new work, but also for the fact that he is an original research man of the most pronounced and practical type, namely, that his research work tends to a practical application, that he is following a most characteristic American style, that is, of applying things in a way in which they stick. I have the pleasure of introducing to you Dr. George W. Crile, of Cleveland.

DR. CRILE: I have paid many visits to the clinic of Dr. Murphy, and I find that its attractions increase from year to year. We have all been pupils of Dr. Murphy. I have been his pupil ever since I have been in medicine, and I expect to be his pupil all my life. I am trying continually to use the things he shows—to apply his methods.

I noticed this morning that the Sister gave nitrous oxid anesthesia skilfully. This suggests my saying a word about this anesthetic. Nitrous oxid skilfully given, in my estimation, is the best anesthetic for surgical purposes, but it ought never to be given except by some one who has been well trained in its administration. In my judgment, the degree of M.D. does not qualify any man to give nitrous oxid anesthesia or any anesthetic, in fact. It gives any one a *legal* qualification, but it does not give one a *moral* qualification. It takes a special training to give nitrous oxid, for it is the most difficult of all anesthetics to give well.

Now, gentlemen, I am speaking about this because I have known of certain fatalities from nitrous oxid anesthesia administered by inexperienced anesthetists. I believe that when well

given, it is the safest anesthesia one can use, and I think that nitrous oxid not well given is perhaps the most dangerous anesthetic there is. Now, in my own clinic in the Lakeside Hospital we have given nitrous oxid for general surgical purposes 8000 times. I myself spent a great deal of time some years ago in working out the nitrous oxid problem in my laboratory. I watched the progress of this anesthetic, and I took especial pains to train our first anesthetist, who in turn now trains others. It takes six months for a good training in nitrous oxid administration. I would not for one moment consider myself competent to give nitrous oxid to any patient, for I believe that it requires a particular training of the reflexes to give this anesthesia well.

In conclusion I can only say, as I said before, that I have visited this clinic many times, and always with profit. It seems that Dr. Murphy never gets to the bottom of the well of his knowledge. I am sure that every one here joins me in the hope that the influence on the surgery of America and on the surgery of the world which emanates from this clinic will flow on for many years to come.

METASTATIC INFECTIONS

A Talk by Dr. George Emerson Brewer, of New York,
at the Clinic, held on Friday, November 14, 1913

DR. MURPHY: While I am doing this preparatory work, I will call on Dr. Brewer to talk to you on any subject he desires. I know from hearing him that he can keep right up all day and the next day if necessary. Gentlemen, I take pleasure in introducing to you Dr. George E. Brewer, of New York.

DR. BREWER: Gentlemen: I think you all appreciate what it is to be suddenly called upon to speak to an audience of this kind, particularly after such a splendid exhibition of surgical teaching as we have witnessed this morning. I feel you will all agree with me that, of all the forceful lessons we have learned in Chicago this week, illustrated by many skilful operations in the various clinics, the one emphasized by Dr. Murphy this morning, and illustrated by his advanced surgical work in the treatment of metastatic infections, particularly in the joints and bones, will be regarded as the most important. Nothing has impressed me more than the very forceful and emphatic manner in which Dr. Murphy has shown us that in these acute metastatic septic accidents occurring in bones and joints, or, in fact, in any part of the body, the greatest danger is delay. These lesions are essentially destructive lesions, and, if we procrastinate, or content ourselves with watching our patient, we assume the gravest responsibility. Dr. Murphy has shown us that in an acute metastatic infection of the joint destruction of that joint takes place with great rapidity if the process is allowed to go on without relief. The same is true in infection of bone, as an early operation which relieves tension and allows the escape of the products of infection will do more to bring about a cure and complete restoration of function than a far more extensive operation at a later period. The same

is true of the metastatic infections which occur in the kidney. These often are extremely virulent, and if unrecognized and untreated, either lead to the death of the individual from an overwhelming toxemia, or go on to the complete destruction of the organ by a purulent infiltration—necrosis; or one of the easily recognized suppurative lesions, as pyonephrosis, pyelonephritis, and perinephritis. The recognition, at the earliest period, of these infections and of the nature of the lesions, with prompt operative interference, often results in a rapid recovery and saving of an important organ. At a later period nephrectomy is often required.

I will not take more of your time this morning, but will say in conclusion that I have been enormously impressed with the magnificent amount of material which we have seen during this session of our Society; but the lesson I personally have learned has been a more thorough understanding of the importance of recognizing in the early stage these destructive metastatic lesions of infective origin.

GASTRIC ULCER AND GASTRIC CARCINOMA.
GASTRIC ANALYSES ILLUSTRATING VARIOUS TYPES OF THESE LESIONS, BY MR. HERBERT PATERSON, F.R.C.S., OF LONDON, ENGLAND

A Talk Delivered at Dr. Murphy's Clinic at Mercy Hospital by Mr. Paterson, on Friday, November 14, 1913

DR. MURPHY: While I am preparing the case for demonstration, I have the pleasure of presenting to you Mr. Herbert Paterson, of London, who will give us some useful information from his deductions and his experiences in connection with the work on duodenal and stomach lesions. This very instructive work Mr. Paterson has done has been one of the lights that lead out of the wilderness.

MR. PATERSON: Dr. Murphy and Gentlemen: I do not think I ought to talk about gastrojejunostomy, because my friend, Sir Arbuthnot Lane, has told us that it is an obsolete operation and therefore it cannot be of interest. I asked him for a definition of this obsolete operation, but receiving no answer, I will give you one myself. An obsolete operation in these cases is an operation that we cannot do without! I should like, first of all, to say a few words on a subject which has been interesting me very much. I refer to the grafting of malignant disease on simple ulcer of the stomach. As you are aware, the Mayos teach that 71 per cent. of cancers of the stomach have their origin in simple ulcer. I am very skeptical as to the accuracy of this conclusion. In doubting its accuracy I feel sure that you will not think I am lacking in appreciation of the work done at the Mayo Clinic, because I, in common with all who have seen the work done there, have the highest admiration for it.

Now as to the reasons for my skepticism. The first argument in favor of the great frequency of malignant degeneration of ulcers is that in 71 per cent. of the cases of gastric cancer seen at the Mayo Clinic there is a clinical history of gastric ulcer, often of long duration. Now I would like to know, what is a clinical history of gastric ulcer? I think there is no disease of the abdomen which is more difficult to diagnose than gastric ulcer. In the vast majority of cases you can diagnose duodenal ulcer, but I do not think that you can, with any degree of certainty, diagnose a gastric ulcer. Now that we are getting to know more of the lesions inside the abdomen associated with the supposed symptoms of gastric ulcer, we know that we may operate on a patient who has all the supposed signs of gastric ulcer,—pain, hemorrhage, and vomiting,—and yet when we open the abdomen there is no lesion in the stomach or duodenum. In such cases we may find an appendicitis or a simple intestinal kink, such as is described by Lane. I believe that a clinical history of ulcer is no evidence that an ulcer has existed.

Next, as to the pathologic evidence. It is that, when a supposed simple ulcer is excised, groups of cells resembling carcinoma, surrounded by connective tissue, frequently are found at the margin of the ulcer. I venture to doubt whether any one can say positively whether such small isolated groups of cells are innocent or malignant. But I will not press this point. For the sake of argument I will assume that it is possible to say that such cells are malignant. Then my answer is this: Supposing there is an ulcer history going back for four or six, or even eight or nine, years, what evidence have we that the ulcer was not malignant from the first? Can any one deny this possibility? Until we know more of the life history of cancer, we are not in a position to deny that a gastric cancer may exist for years. Let me give a case in support of this contention. In 1907 I opened the abdomen of a man who had a gastric carcinoma. When I opened the abdomen I found a carcinoma too far advanced for removal. I took sections of it, and we proved definitely that it was a carcinoma. I performed an anterior gastrojejunostomy; I also took away a piece of mucous membrane of the stomach at

the site of the anastomosis, and found nodules of carcinoma cells in that part of the stomach also. Of course, any radical operation was out of the question. That was six years ago; and at the present time that man is perfectly well. He was ill two years before we saw him, so you have a case of carcinoma of the stomach of at least nine years' duration. I think that this case shows that we do not know very much about the clinical history of carcinoma, so when you say a patient has an ulcer history of seven or eight years, I think it is very difficult for you to say whether the symptoms are due to malignant disease or to simple ulcer, if, indeed, they are due at all to a lesion of the stomach.

Then there is another very significant feature of these cases. It is not at all uncommon, when operating for carcinoma of the stomach, to find a healed gastric ulcer some little distance away from the cancer. This is just what we should expect would happen sometimes, because in England, at any rate, cancer of the stomach is a very common disease. These patients then have a carcinoma plus an ulcer, and consequently give a history of ulcer as well as a history of cancer, but from what we see on the operating table we know that the cancer and the ulcer are independent. This illustrates the fallacy of the history argument in determining the frequency of the grafting of malignancy on simple ulcers. I do not, of course, deny that such an event takes place sometimes; it is the great frequency of this occurrence which I am disputing. Then there is another argument—and it seems to me a very strong argument—against the view that malignant degeneration of ulcers is a very frequent occurrence. Duodenal ulcer, as American surgeons have taught us, is a more common disease than gastric ulcer, and occurs almost always in the first part of the duodenum. Ulcer of the second part of the duodenum is very rare. What about carcinoma of the duodenum? Now carcinoma of the duodenum is a very rare disease, and when it does occur, it is met with, not in the first, but in the second, part of the duodenum. If carcinomatous degeneration of ulcers occurs in 71 per cent. of gastric ulcers, why is it that hardly ever do we see a carcinomatous degeneration of an ulcer of the first part of the duodenum? I think that is a tough proposition to face.

Then I have another argument. If carcinoma develops frequently in gastric ulcer, then it must often happen that we perform a gastrojejunostomy on patients who have simple ulcers, but which are already carcinomatous. Consequently a considerable number of patients on whom we perform gastrojejunostomy for simple ulcer ought to die subsequently from malignant disease. Is this the case? There can be only one answer: It is not the case.

Dr. Gressot followed up a large number of cases, and found that after gastrojejunostomy for simple ulcer less than 3 per cent. of the patients died of carcinoma. Professor Kocher followed his cases back for fifteen years, and found not a single patient died of carcinoma. My own experience is very much the same. I have seen only two cases of patients who died from carcinoma after a gastrojejunostomy for supposed simple ulcer. In both of these patients I was able to get a postmortem and found the carcinoma originated quite a distance from the original ulcer for which I performed the gastrojejunostomy, so I think we still require additional evidence before we accept the conclusion that in 71 per cent. of gastric cancers the disease originates in a simple ulcer of the stomach.

Next I will show you a few slides illustrating the value of gastric analysis in diseases of the stomach. Many believe that gastric analysis is no good. I am one of those who find it a very great aid in diagnosis. I think no surgeon can afford to do without its help. At the same time, I would point out that gastric analysis is an aid to, but not a substitute for, clinical observation. If we trust to it entirely, we will be misled; if we use it as an adjunct to the history of the patient, I think we will be greatly benefited.

GASTRIC ANALYSIS

Duodenal Ulcer

Total acidity.....	80
Total chlorids.....	0.405
Free HCl.....	0.055
Protein HCl.....	0.275
Mineral chlorids.....	0.075

This is a very typical analysis from a case of duodenal ulcer. If you get an analysis of this kind in conjunction with a history of duodenal ulcer, there is no question that the patient is suffering from a duodenal ulcer. I never make an absolute diagnosis of duodenal ulcer unless the patient has a typical analysis. I know of no condition in which you get an analysis like this except in association with a duodenal ulcer or a gastric ulcer close to the pylorus. You will observe the points are these: a high total acidity of 80; a very large percentage of total chlorids—nearly double the normal; a very high free hydrochloric acid; protein hydrochloric acid fairly high; mineral hydrochloric acid about normal.

GASTRIC ANALYSIS
Duodenal Ulcer

Total acidity	78
Total chlorids	0.335
Free HCl	0.000
Protein HCl	0.235
Mineral chlorids	0.120

Here is another analysis of a case of duodenal ulcer. This is an analysis in a kind of case in which there is often considerable difficulty in making a diagnosis. You will note there is no free hydrochloric acid present. When you get free hydrochloric acid absent in the analysis, one is always chary of making a diagnosis of duodenal ulcer. There are several conditions associated with duodenal ulcer which lead to the absence of free hydrochloric acid. This often happens if you have a duodenal ulcer associated with gall-stones. Free hydrochloric acid may be absent also if the ulcer has healed, or if the ulcer has become contracted so as to cause pyloric stenosis. It also happens when you get a duodenal ulcer associated with an intestinal stasis or with an appendicitis. This patient had a gall-stone and also an ulcer which had produced stenosis.

GASTRIC ANALYSIS

Gastric Ulcer (Close to Pylorus)

Total acidity.....	75
Total chlorids.....	0.325
Free HCl.....	0.040
Protein HCl.....	0.205
Mineral chlorids.....	0.080

This is an analysis from a case of gastric ulcer close to the pylorus. You will notice that the analysis is almost identical with the analysis of a duodenal ulcer. I think it is quite impossible to distinguish gastric ulcer close to the pylorus from duodenal ulcer. The analyses are similar and the clinical histories are almost identical.

GASTRIC ANALYSIS

Gastric Ulcer (Middle of Stomach)

Total acidity.....	78
Total chlorids.....	0.308
Free HCl.....	0.025
Protein HCl.....	0.206
Mineral chlorids.....	0.077

Here is an analysis of a case of gastric ulcer in the middle of the stomach. You will observe that the free hydrochloric acid is only slightly above normal; the total chlorids less than in the duodenal ulcer; the protein hydrochloric acid less than in the duodenal ulcer.

GASTRIC ANALYSIS

Gastric Ulcer (Hour-glass Stomach)

Total acidity.....	70
Total chlorids.....	0.332
Free HCl.....	0.000
Protein HCl.....	0.230
Mineral chlorids.....	0.098

This is an analysis taken from a case of hour-glass stomach. A very important point about hour-glass stomach is that there is invariably an absence of free hydrochloric acid. Another

point of importance is that often a patient with hour-glass stomach is extremely emaciated, so that when an analysis is made and no free hydrochloric acid is found, a diagnosis of carcinoma is made and no operation is performed. It is important to remember that there is no free hydrochloric acid present in hour-glass stomach. If you suspect the condition, it is quite easy to diagnose it by other methods. You will notice that that analysis differs slightly from that of a carcinoma. The total acidity is fairly high; the total chlorids are fairly high. You will see the total acidity in a carcinoma is rarely as high as this and that the total chlorid is rarely as high as 202

GASTRIC ANALYSIS

Appendicular Gastralgia (Intestinal Stasis)

Total acidity.....	52
Total chlorids.....	0.250
Free HCl.....	0.000
Protein HCl.....	0.167
Mineral chlorids.....	0.083

After Appendicectomy

Total acidity.....	72
Total chlorids.....	0.368
Free HCl.....	0.014
Protein HCl.....	0.270
Mineral chlorids.....	0.083

Here is an analysis for a case of intestinal stasis. In this instance the intestinal stasis was associated with appendicular disease. You will notice there is almost a complete absence of free hydrochloric acid; a normal total acidity; a nearly normal total chlorid. An interesting point about these cases of intestinal stasis is the absence of free hydrochloric acid. To what is this absence of the hydrochloric acid due? I used to think it was due to a pyloric spasm, but on examining the patient with the x-ray one found there was no pyloric spasm. I tried stimulating the appendix during operation, and found that this had no effect on the pylorus.

There is an interesting point about this absence of free hydro-

chloric acid to which I called attention several years ago, namely, that after operation, not within a few months, but within a few days, one finds a restoration of the free hydrochloric acid in the gastric contents. I think that there is only one possible explanation of this, and that is that in intestinal stasis there is an absorption of some toxin from the intestinal canal, which directly interferes with the secretion of free hydrochloric acid in the stomach. There is one point which I should have mentioned in connection with hour-glass stomach, and that is that as the oxyntic cells are at the cardiac end of the stomach it is curious that there should be an absence of free hydrochloric acid. I think the explanation is that as the food cannot pass readily into the pyloric end of the stomach, there is inadequate secretion of the hormone which stimulates the oxyntic cells. In intestinal stasis the secretion of the oxyntic cells may be inhibited directly by some toxin, or the secretion of the hormone which stimulates the oxyntic cells may be inhibited.

GASTRIC ANALYSIS

Gastric Carcinoma

Total acidity.....	28
Total chlorids.....	0.178
Free HCl.....	0.000
Protein HCl.....	0.051
Mineral chlorids.....	0.124

This is an analysis from a case of gastric carcinoma. You will observe a very low total acidity—only 28; total chlorids under 200; complete absence of free hydrochloric acid. In my opinion the most important of all is that there is a very low combined or protein hydrochloric acid. When you get an analysis like this, it is always suspicious of a carcinoma.

Here is a photograph from a case of hour-glass stomach which is very interesting from this point of view. On looking on the outside of the stomach you would not think there was much the matter with it, but on examining the inside of the stomach one finds there is a marked hour-glass stomach. The important point to remember is that many cases of hour-glass stomach are



Fig. 44.



Fig. 45.



Fig. 46.

overlooked because there is so little indication outside of the serious condition within.

Here is a large duodenal ulcer which has become adherent to the liver, so that the base of the ulcer is formed by liver. Constant pain radiating through to the back is suggestive of involvement of the peritoneum over the ulcer or of surrounding viscera.

GASTRIC ANALYSIS

Chronic Gastritis

Total acidity	28
Total chlorids	0.178
Free HCl	0.000
Protein HCl	0.051
Mineral chlorids	0.124

Here is an analysis from a case of chronic gastritis. I want you to compare it with the next analysis. You will observe the low total acidity; the low total chlorids; the absence of free hydrochloric acid; and the protein hydrochloric acid.

GASTRIC ANALYSIS

Gastric Carcinoma

Total acidity	29
Total chlorids	0.183
Free HCl	0.000
Protein HCl	0.070
Mineral chlorids	0.113

Now here is another analysis which is very similar to the one I have just shown you. The point is how to differentiate between these two analyses and to distinguish which is carcinoma and which is not.

The first analysis is from a chronic gastritis, and the second from a case of gastric carcinoma. I will tell you about the two cases.

The first analysis is from a man aged thirty-five years, who had been ill for twelve months with gastric symptoms. The analysis shows a marked degree of gastritis. Notwithstanding the duration of his illness he was in fairly "good shape," was not

anemic, and had lost little weight. You will notice that the total acidity is low, although not so low as in the cancer case; there is no free hydrochloric acid, and the total chlorids are greatly diminished. The analysis, taken by itself, suggests carcinoma, but interpreted in the light of the clinical history, the general well-being of the patient, considering the length of his illness, one had no difficulty in coming to the conclusion that he was suffering from a chronic gastritis of inflammatory origin. At the operation a gumma of the liver was found which had become adherent to the pylorus and produced stenosis. A gastrojejunostomy was performed and was followed by complete relief.

The second analysis is from a woman who gave a history of gastric trouble of three months' duration. She had slight anemia and had lost weight. The extreme degree of gastritis shown by gastric analysis, coupled with the short duration of the symptoms, suggested a diagnosis of carcinoma, which was confirmed by operation; the growth, which was at the pylorus, was successfully removed, and the patient is now alive and well three years after the operation. I do not think that gastrojejunostomy is yet an obsolete operation. In proper cases the results are excellent. I think we ought not to forget how much we owe to Dr. Murphy for his ingenious button, for it was this invention which gave the stimulus to intestinal surgery which has made gastrojejunostomy such a safe operation. Originally gastrojejunostomy was always performed on the anterior wall of the stomach. Then the posterior operation was introduced, and for a long time there was a fierce contest as to the relative merits of the two operations. After a time it was decided by some authorities that the only operation to be done is the posterior operation. The man who performed the anterior operation was a man past praying for. I confess that latterly I have become one of the renegades. I am not certain that the anterior operation is not so good as, if not a little better than, the posterior. I do not think there is any reason why the anterior operation should be followed by regurgitation of bile any more than the posterior. At the time the Murphy button was used the an-

terior method was adopted invariably, and I think it was the experience of all who used the button that regurgitant vomiting was exceedingly rare. Be this as it may, my belief is that the type of operation employed is of less importance than the manner in which it is done. Lastly, I should like to express the pleasure I feel at being in America once more, and to thank Dr. Murphy and you, gentlemen, for the kind manner in which you have listened to my remarks.

DR. MURPHY: We feel very much indebted to Mr. Paterson for the presentation of this subject of gastric analysis. That has added some additional elements and positive views to the differential diagnosis from the laboratory side. We are agreed with what Mr. Paterson so beautifully demonstrated, that all these things are but aids to the clinical course and clinical history, to confirm the clinical course and the clinical history in that way. These aid us in arriving at a decided diagnosis, that is, a patient-made diagnosis and not at a machine-made diagnosis; that is, the patient, his symptoms, and his clinical course, with these laboratory additional things applied, aid us in coming to a positive conclusion, more definite and more frequently correct than we otherwise would.

UNUNITED FRACTURE OF THE ULNA. TRANS- PLANTATION OF BONE FROM TIBIA

HISTORY

PATIENT, female, aged nineteen, entered the hospital November 7, 1913, because of a deformity and false point of motion in the upper one-third of the left forearm. The hand is held slightly pronated, and the patient is unable to supinate it.

The present trouble dates back to two years ago, October, 1911. At that time the patient was driving a horse, which became balky and kicked over the dash-board, striking her on the left forearm. The horse then ran away, dragging her from her seat in the buggy onto the ground. Immediately after the injury she had severe pain in the left forearm, and on inspection she noticed some deformity. On arriving home the arm was swollen. She did not try to move it because of the pain. A doctor was called, and he placed a splint on the anterior surface of the forearm, securing it in place with rollers. This splint was left on for seven weeks, although it was removed several times and the arm examined fluoroscopically.

After seven weeks the splint was removed permanently and the patient was told that the arm was in perfect condition. The patient noticed, however, that she could not flex her elbow. Therefore, when there was no improvement in the range of motion in the elbow she went back to the doctor after three weeks. He told her then—after he examined the arm—that the bones had slipped. She was taken to the hospital, an incision was made over the ulna, the fragments were approximated, and a Lane plate put on and secured with two screws. No splint was applied this time. The arm was merely bandaged. The wound healed in about ten days, but the patient still was unable to flex her forearm.

Ten months later (November, 1912) she consulted another doctor. He made a skiagram and found that the head of the radius was luxated, and that one screw in the Lane plate was loose. One year ago (November, 1912) the plate was removed. The head of the radius was removed and a piece of this bone was transplanted into the gap between the two ends of the ulna. The arm was placed at right angles against the body and held there by means of casts and splints, extending from the metacarpophalangeal joints to the middle of the upper arm. During the last six weeks she has had nothing on the arm, and it was not until the casts and splints were removed that she noticed the deformity of the forearm and inability to supinate it.

COMMENTS AND OPERATION

DR. MURPHY (Operation November 12, 1913): That is not quite a typical history. That case has some features that are not common. One is in the fact that the upper end of the radius has been removed. It is now a case of non-union of the ulna. There is complete motion in the elbow-joint, which is very fortunate, but this woman is practically incapacitated because of the false point of motion in her forearm. If she had an ankylosis of the elbow, then the condition would be more serious than it is now. You will note in the skiagrams (Figs. 47, 48) that the upper portion of the radius is gone entirely, and yet the patient has the power of flexion of the forearm were it not for the flail joint in the ulna, 3 or $3\frac{1}{2}$ inches below the elbow, which prevents her from doing anything with the forearm. Just how the biceps is situated in that case I do not know, but it is probably a part of the general scar tissue where the radius was removed, as the radius was probably taken out subperiosteally, and that is what gives the flexion strength. In the attachment of the tendon of the patella it is the fascia that spreads out that carries much of the weight, and gives very much of the strength to the tendon patellæ attachment.

The question is, how are we going to handle this case? It is an ununited fracture in the upper one-third of the ulna—not quite as common as an ununited fracture in the lower third of the



Fig. 47.—Fracture in upper portion of ulna and radius, with non-union. The head of the radius was removed at a previous operation (lateral view).



Fig. 48.—Anteroposterior view. The non-union shows well in this picture. The radius and ulna are united transversely.

humerus, which is one of the most common positions of fracture. The next most common type is the ununited fracture of the upper third of the humerus; then the ununited fractures of the lower third of the forearm; then the ununited fractures of the lower third of the leg; then the ununited fractures of the upper third of the leg, and then the femur. That is about the order of frequency of non-union of fractures.

Non-union of fractures has become a very much more common condition than it was formerly. Just why that is so has not yet been determined definitely. It is my opinion, however, that many of the cases of non-union are the sequences of a too perfect immobilization following fractures. In other words, that to stimulate osteogenesis it is essential that there should be some motion at the line of fracture. It is also essential that there should not be too much motion at the line of fracture when this is near a joint. That, however, is another matter. There are some other elements which we cannot explain, but the clinical fact remains that non-union occurs, and when it does occur, what are we to do about it?

Formerly we freshened the surfaces of the fragments, brought the ends together and wired them, and we had a certain percentage of union. Then we changed from that to the method of plating, and there we had a better percentage of union, but in all these methods of fixation there was a definite large percentage of failure of union. It was to meet that condition that we many years ago resorted to bone transplanting for the purpose of insuring a union. In this line of work we have been very well pleased with the final results. There are, however, definite things to be done in an exact and definite way if one is to meet with uniform success in that work. One of the greatest determining factors in securing good results in that class of cases is *absolute asepsis*, and that asepsis is much more difficult to attain in bone and joint work than it is in abdominal work—but it can be attained.

We do not always put in a medullary bone splint or transplant when we have a non-union. The skiagrams of some of our early cases show that we have used subperiosteal splints, auto-

bone transplants placed on the surface of the fragments and fastened there by catgut or phosphor-bronze wire wrapped around the transplant and the bone itself. A medullary splint requires the smallest quantity of bone which gives sufficient strength to hold the fragments in place.

I said that in order to avoid non-union in fractures not near a joint it was essential to have a limited motion. In order to insure a union after bone transplantation it is absolutely necessary to have motion restricted to the greatest possible degree—in other words, absolute immobilization. Why these two different considerations? I may be in error as to my theory of it, but I am not in error as to the results. Non-union is the result of insufficient stimulation to produce the action of the osteoblasts. In the case of the transplant, what is the difference between these requirements? In the primary union of bone osteoporosis occurs back of the line of fracture; then there comes the formation of the granulation tissue; next, the formation of the fibrous callus; next, the transmission through that fibrous callus of the blood-vessels carrying the osteoblasts. If there is too much motion, there is developed over the end of that bone a connective-tissue mass, and the result is a failure of union, just the same as occurs in the regeneration of a divided nerve. If you unite the bulbous ends of a divided nerve without axonal contacting, failure of axonal union and failure of subsequent transmission of impulses are the result.

In the transplantation of bone it is essential that the osteogenic elements, the osteoblasts and the osteoclasts, and the Haversian vessels of the living fragments above and below the line of fracture, pass into and through the Haversian canals of the transplant. If the rôle of the transplant is to be a mechanical one, immobilization to a superlative degree must be secured. The transplant becomes osteoconductive. The Haversian vessels with their osteoblasts and osteoclasts pass into the Haversian canals of the transplant, the osteoblasts depositing cells and the osteoclasts removing cells. New bone replaces the dead bone, and a network of new bony trabeculæ is formed long before all the old bone is removed by absorption. It is my belief that most, if

not all, of the old bone finally disappears, but that does not matter to us as surgeons. If, however, you do not immobilize the transplant, that connective-tissue mass forms around the transplant and the Haversian vessels do not get into it from the fractured ends and a non-union results. I want to make that clear because these are two distinct and clear clinical conditions.

As far as the material to be used for transplanting is concerned, we have been reading very much lately of "cold storage" transplants and "cold storage" preservation of kidneys and other organs, and the transplantation of these tissues with or without supports. They are not of permanent value in transplantation.

In order to achieve the best results in the work of bone-grafting and bone transplantation, the following laws must be observed:

I. The periosteum fully detached from bone and—(1) Transplanted into a fatty or muscle-tissue bed in the same individual, if he be young, may produce a lasting bone deposit; (2) transplanted into another individual or animal of the same species and under the same conditions, it rarely, if ever, produces a permanent bone deposit; (3) transplanted into another species it never produces a permanent bone deposit.

II. Periosteal strips elevated at one end from the bone and attached at the other, if turned out into muscle or fat, reproduce regularly bone on their under surface for a greater or lesser portion of their entire length.

III. Transplanted into other individuals or animals of same species and contacting at one end with exposed or freshened bone, it rarely produces permanent bone, even for a small extent at its basal attachment, and never produces bone for its full length.

IV. Bone with its periosteum transplanted into muscle, fat, etc., in the same individual, and free from bony contact, practically always dies and is absorbed, except in the case of very young children or infants. Transplanted into another species it is always absorbed.

V. Bone transplanted without the periosteum into the muscle or cellular tissue always dies and is ultimately absorbed.

VI. Bone with or without periosteum transplanted in the

same individual and contacted with other living osteogenetic bone at one or both of the ends of the transplanted fragment always becomes united to the living fragments and acts as a scaffolding for the reproduction of new bone of the same size and shape as the transplanted fragment if asepsis is attained. This new bone increases to such size as is necessary to give the support required by nature in the extremity in which it has been placed. It will scaffold the production of new bone even into the joint when it is surrounded by capsule, and tuberosities are produced in about the regular location, as in the normal anatomic conformation if the muscles are sutured to the graft.

VII. The transplanted fragment, no matter how large or how small, is always ultimately absorbed. The rôle it plays is to give mechanical support to the capillaries and blood-vessels with their living osteogenetic cells, as they advance from the living bone at both ends of the transplanted fragment into the Haversian canals, canaliculi, and lacunæ of the transplant. New lamellæ are deposited around the new capillaries, and these lamellæ fit into and adjust themselves in the graft, so that the bony union is actually formed and mechanical support given long before the transplant is entirely absorbed and replaced by new bone. Ultimately all of the transplant disappears as new lamellæ are formed by the osteoblasts, and the graft lamellæ are removed by the osteoclasts.

This conclusion is based on my observations and on analysis of the microphotographs and pathologic specimens, clinical and experimental, presented by others. The graft is *per se* not *osteogenetic*, but *osteoconductive*. The regenerative force and cells are entirely supplied from the osteogenetic cells on the capillaries growing from the living bone. The graft, however, is an absolute necessity in the regeneration.

VIII. The graft increases in size on the surface as bone increases in size histologically, *i. e.*, by deposits beneath its newly formed periosteum.

IX. The muscles should be fixed or directed to the graft in the same relation as the normal anatomic fixation, if the muscular control is expected after the graft has become united, and the

musculotendinous attachments should be accurately sutured to the graft at the point of desired union, either with catgut or phosphor-bronze wire.

X. Bone covered at the ends by cartilage and on the sides by periosteum, such as the phalangeal bones, even when contacting with living bone denuded of periosteum, dies and is wholly absorbed. When the graft is covered with periosteum at the point of contact with the living bone, the Haversian vessels do not penetrate the fibrous periosteum of the transplant, and regeneration fails. If the periosteum of the graft is split into shreds, then regeneration through it may take place.

XI. Periosteum attached to the transplanted portion, if the graft is taken from young individuals, has a plus osteogenetic influence; in the middle-aged it is neutral; in those of advanced years it plays a minus rôle, and, in fact, it is detrimental.

While these theories may finally be found defective in some of their details, they represent my observations in the experimental and clinical work I have carried on. Bone reproduction, from a practical standpoint, is permanently and for all time settled both as to its feasibility and its practicability, and the extent to which it can be carried out to meet the mechanical conditions essential to its evolution.

The indications for bone transplantation are:

A. To correct deformities resulting from defects of development, as aplasic extremital bones—radius, ulna, humerus, tibia, fibula, and femur, and congenital saddle-nose, aplasic mandible, etc.

B. To produce union in ununited fractures.

C. To replace bone removed by destructive infections, osteomyelitis, tuberculosis, lues, etc..

D. To restore or supplant fragments dislodged or destroyed by fractures, as the head of the humerus, head of femur, shaft of tibia, etc.

E. To replace bone removed for non-malignant neoplasms, cysts, myeloma, osteitis fibrosa, etc.

F. To replace bone removed for encapsulated malignant disease, as giant-cell and chondrosarcoma, etc.

In the grafting and transplanting of organs and tissues no one has had a larger experience than Professor Tuffier, of Paris. His experience has been mostly with the ovary. The ovary, as you know, is made up of epithelial cells, and epithelial cell structures should be the easiest to transplant, as we know from the grafting of fruit-trees. In the human, when the ovary is transplanted from within the pelvis onto the peritoneum of the anterior abdominal wall beneath the posterior rectus aponeurosis, or when it is transplanted external to the rectus muscle beneath the deep fascia and fat, if it is taken from the same patient and transplanted to either of these positions and in an aseptic way, it always lives.

Professor Tuffier, believing that the patients who had both ovaries removed and who were suffering from the neuroses usual to these cases could have their function of menstruation restored by the transplanting of the ovary from one individual to another, made some clinical experiments. So in operating on a patient whose ovaries were usable for this purpose, he would remove the two ovaries, put one in the same patient and the other into the patient who had lost both ovaries. The one that was transplanted in the same patient to another position always lived. The one that was transplanted to another patient always died. I had made that statement before with a little reservation, because when I saw Professor Tuffier two years ago he said he thought one ovary would survive, but in this clinic last week he said he never had one survive. Then he tried transplanting an ovary from a black to a white woman and from a white to a black woman. The result was a failure. All the homotransplants perished. All the autotransplants lived if aseptically transplanted.

That is exactly in consonance with our observations in transplantation of bone. Bone taken from the same individual and transplanted to another position in his body always lives. All these grafts have lived in our work. We have not lost a single bone transplant. Some of the men who may be here today heard me state about a year ago that I had one case in which I felt I was going to lose the graft, but eventually only a little piece

the size of a darning-needle came out. The remainder of the graft lived, and our 100 per cent. of success was again a fact. I operated on that case two weeks after the boy had the grip. He got a pneumococcus infection in his leg. He was treated with vaccines, and notwithstanding that he had a suppuration around the upper end of the graft, the graft lived, all but a little needle-shaped piece at the upper end. We had another case where a little piece of the transplant was removed from a forearm.

You will see in the case before us today that I have but very little space in which to put the upper end of the graft, because of the shortness of the fragment. I have plenty of space for the lower end. There is a considerable displacement of the ulna. There is a real false joint, a pseudo-arthritis, which I will endeavor to demonstrate to you.

I will expose the ulna through an incision on the posterior surface of the arm, just to the outer side of the ulna, so that the scar will not lie over the crest of the ulna subsequently. Retracting the wound edges, the fragments come into view. We must remove the soft tissue between the fragments and freshen the ends of the bones. Towels are placed on the edges of the wound to keep from contacting with the skin. I will keep behind and beneath this bone and its periosteum. I am making a dissection and removing the anconeus, so as to have an exposure of the upper fragment. There is the pseudo-arthritis,—a typical pseudo-arthritis,—with the development of a condition closely resembling a normal joint. The observations on cases of this class led us originally to do our arthroplasty work in the construction of new joints.

Now the ends of both fragments are coming into the field. It was said that a portion of the radius was transplanted between both ends of the ulna, but it does not show in this specimen. The new joint tissue resembles synovial membrane—a soft fibrous layer. All of that must be taken out. Why? So that the osteogenetic forces will pass from the living bone into the transplant. Remember that is a very essential step in this work—to establish conditions which will allow the osteoblasts

and osteoclasts to make their way into the Haversian canals of the transplant. The fragments are fully exposed now in their entire circumference. I have removed the tissue that resembled the new articular surface from the end of the ulna, so I will not have any interposing soft tissue to prevent subsequent union between the fragments. In non-union the development of a connective-tissue capsule over the ends of the fragments has been the greatest detriment to the union of bone. It has always been one of the greatest drawbacks. That end is ready, except for the drilling. We always ream out the medullary canal so that we can implant a fragment of bone of sufficient diameter to give a good support to the arm until the new bone has formed. We ream out the lower fragment also to a depth of about $1\frac{1}{2}$ inches.

There is the radius. We wish the radius to stay united to the ulna in this position, because it is a good fixation. The piece of bone that can be implanted here must be comparatively small, as the lumen of this bone is small. It is not the size of the transplant so much that counts as it is the fact that it is conductive of osteogenetic elements. That is why it is valuable.

A medullary graft gives the most perfect immobilization, and perfect immobilization is essential to repair.

We always take the graft, where possible, from the crest of the patient's tibia. We cut the skin not directly over the crest of the tibia, but a little to the outer side, over the tibialis anticus muscle. We split the aponeurosis of the muscle close to the bone, retract the muscle, and then divide the periosteum. We always take the periosteum with the transplant. Why? Because we have always done so, and inasmuch as we have had 100 per cent. of success, why should we change our technic? We do not believe that the periosteum is a necessity—it is merely an advantage, although in one of our cases it was a detriment. In driving the transplant into the ends of the fragments the periosteum is undoubtedly stripped off the bone, but we pay absolutely no attention to this. We have done all our transplantations in this way and so far we have not had a single failure.

We always remove the transplant with a small buzz-saw.

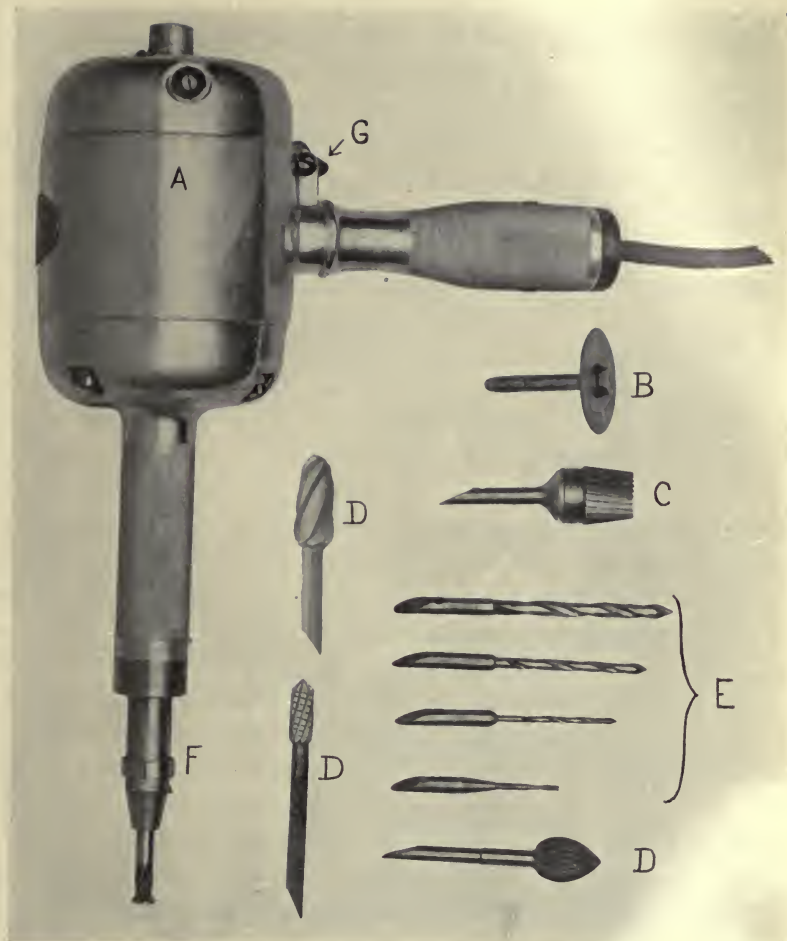


Fig. 49.—Dr. Charles A. Geiger's electric motor, bone-saws, and drills. The motor, A, is part of the hand-piece, and can be sterilized. It is intended to supplant the cable, which usually conveys the power from the motor to the instrument. The saw B, the trephine C, the reamer D, and the drills E, are fitted into the chuck F. The little thumb-piece G starts and stops the motor. The entire apparatus weighs four pounds.

It saves time and energy. We will remove a piece of bone about three inches long by $\frac{3}{8} \times \frac{5}{16}$ inch. It is done very rapidly. The apparatus we are using this morning was devised by Dr. Charles Geiger, of St. Joseph, Mo. The motor is in the handle of the instrument. It works very well, indeed. (See Fig. 49.)

You see it is a small transplant, but it is too long, so I cut off about one inch, saving the piece in case I need it later. Now I am ready to place the transplant into the fragments. I first drive it down into the lower fragment and then it is placed into the upper fragment. By catching the edge of the graft with the sequestrum forceps I can drive it back into the upper fragment and have it secure. Now that arm is firm.

I am going to make an external graft of the remaining portion of that transplant. I will put it on the surface of the fragments, and on the inner side, first removing the periosteum so that bone will contact with bone and this piece will pass directly across the line of union.

In order to get a union the transplant must contact with living bone at both ends or laterally with both fragments. All we are doing with that is to immobilize it with catgut. You see there is a contacting of two freshened bony surfaces here. If I permit the periosteum to be interposed between these two fragments, there would be no regeneration of bone and that fragment would finally be absorbed. Three catgut sutures are put in to press that bone against the surface of the fragments until such time as they unite.

The wound is closed with horsehair, dusted with bismuth subiodid, and sealed with collodion gauze. An alcohol-phenol gauze dressing is applied, and the arm placed in a metal circular splint covered with leather, extending from wrist to shoulder, with the arm at a right angle flexion and the hand midway between supination and pronation. The wound in the leg is closed by means of catgut, which approximates the edges of the muscle aponeurosis and horsehair for the skin incision. The same dressing is applied as in the arm, except for the cast.

EXHIBITION OF CASES

Let me show you some lantern-slides of cases of bone transplantation treated in this clinic. The first case was one of fracture of the humerus in which a Lane plate was used. The first picture (Fig. 50) shows the arm as it was when we first saw the case. A good apposition of the fragments,—just as perfect an apposition as it is possible to produce,—and yet not the slightest sign of ossification. That is an interesting matter. It means that you can have an absolute apposition without any ossification occurring. Why? That point has not yet been cleared up, but the clinical fact remains that it is true. As stated before, we believe it is due to too perfect immobilization. That patient came here because of a non-union. He had a pseudo-arthritis. We did a transplantation and put in a nail to keep the fragment from sliding up or down. The result was a splendid one. A firm union occurred (Fig. 51).

In the case operated today a nail was not used; the graft was tied to keep it from moving. You saw what a small intramedullary graft that was, and still we have a perfect alinement.

We now use a leather-covered metal cast or mold in these cases. It can be used on either arm, by adjusting it. It is a good apparatus, just fixes that arm in an easy and comfortable way. It is preferable to a plaster cast. To produce the immobilizing we desire, a cast must be large and cumbersome. This patient was operated on the twenty-second of September, about six weeks ago, for an ununited fracture of the humerus. Note the perfect alinement of his arm as shown in the skiagram (Figs. 52, 53, 54). This is the plan of procedure followed in all our non-union cases. Remember we do not include in the "non-unions" the cases of deformity where there is an attempt at union or even union in a deformed position.

Here is another case of the same class produced in a different way (Figs. 55-61). This was a shot-gun injury of the humerus, with a destruction of the musculospiral nerve and the development of a pseudo-arthritis about three inches above the elbow. He has an ankylosis of the elbow as well. He could not flex his

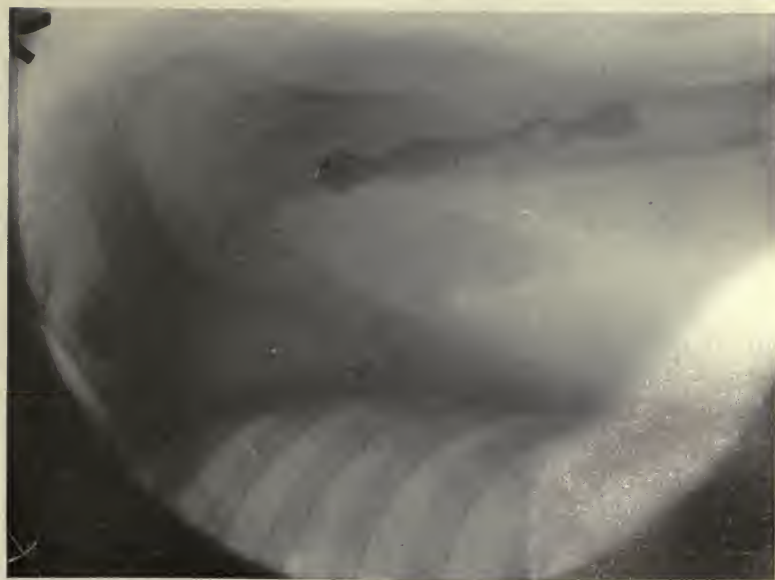


Fig. 50.—Fracture in upper portion of humerus, with Lane plate applied. Perfect apposition of fragments, but no effort at union.



Fig. 51.—Skiagram made four months after transplantation. The arm was solid, and this picture shows considerable osteogenesis. The final result in this case is splendid.



Fig. 52.—Fracture of lower end of humerus, with non-union of fragments. The position at this angle is good, but there is no evidence of osteogenesis.



Fig. 53.—Anteroposterior view. Slight angulation of arm, with apparent union of fragments. Comparing this picture with the previous one emphasizes the necessity of making skiagrams from more than one angle in every case.



Fig. 54.—Lateral view made six weeks after transplantation. The alignment is perfect. The nail is driven through the shaft and the transplant to prevent upward or downward displacement of the latter. The ends of the fragments were squared off with bone-cutting forceps at operation.



Fig. 55.—Fracture of lower end of humerus, with non-union; pseudoarthrosis; musculospiral paralysis due to division of nerve; bony ankylosis of elbow. There is a discharging sinus just above the joint.



Fig. 56.—View of arm from behind, showing the deformity just above the elbow.



Fig. 57.—View of arm from in front.



Fig. 58.—Skiagram, lateral view, from inner aspect of arm, before operation. Bony ankylosis of elbow with joint flexed at right angles. Non-union of fracture with formation of a typical pseudarthrosis. The black spots are remnants of the shot which caused the fracture.



Fig. 59.—Lateral view from external aspect of arm before operation. The normal anatomy of the elbow is here seen to be obliterated completely. The joint is a solid bony mass. From this angle a considerable callus formation on the ends of the fragments is shown.



Fig. 60.—Skiagram made five months after the operation, at which a sliding joint was made at the line of fracture. It functionated fairly well, but to increase the range of motion a second operation was performed later, at which more bone was removed from the anterior surface of the lower end of the upper fragment. The arm is in flexion in this skiagram.



Fig. 61.—Skiagram made five months after first operation. Arm is in extension. (Compare with same view before operation, showing how much more straight the arm is here.)



Fig. 62.—Fracture of tibia and fibula (anteroposterior view). Slight displacement of tibia fragments with apparent union (fibrous). The lower fibula fragment is displaced outward and is widely separated from the upper fragment (before operation).



Fig. 63.—Lateral view. Marked angulation of both bones, the upper fragments pressing the lower fragments downward and forward. There is a considerable callus formation on the lower end of the upper tibia fragment, with a fibrous union with the lower fragment. The fibula is behind the tibia in this picture (before operation).



Fig. 64.—Anteroposterior view. Skiagram made about eighteen months after the transplantation. Firm union has occurred in both bones. The tibia is of normal size and shape. The transplant in it is scarcely visible. In the fibula the transplant was embedded partly in the lower fragment and contacted laterally with the upper fragment. The union here is also firm, and there is no trace of the transplant. The new bone passed through the transplant only, not between the fragments themselves, as may be noted by comparing this figure with the same view made before operation.



Fig. 65.—Lateral view made about eighteen months after operation. The leg is straight, and the tibia is a solid mass of bone. The lower end of the transplant in the lower tibia fragment is just visible. The shadow of the fibula is barely discernible through the tibia.



Fig. 66.—Congenital fracture of lower part of tibia and fibula. A Lane plate was placed on the tibia, but union failed to take place. The screws are loose and one is out, but still in the soft tissues (lateral view).



Fig. 67.—Anteroposterior view. The plate is practically loose, although there was no suppuration; the fragments are not in apposition and there is still a deformity.



Fig. 68.—The previous operation having proved a failure, the Lane plate was removed from the tibia in Copenhagen, and the fragments were wired with two loops of silver wire. The fragments were approximated laterally. The wire cut through the upper fragment; union did not take place after this operation. Note aplastic condition of fibula.



Fig. 69.—Skiagram; anteroposterior view, made a few weeks after Dr. Murphy transplanted two fragments removed from the crest of the opposite tibia. The lower end of each transplant was driven through the epiphysis into the lower fragment of the tibia and fibula respectively. The upper end was approximated laterally and held in place with loops of phosphor-bronze wire. A few loops were also thrown around each transplant and the lower fragments. The leg is straight.



Fig. 70.—Lateral view after Dr. Murphy's operation, showing the splendid position of the fragments and the transplant.

forearm. We could not make a new joint, because there was no tissue in the neighborhood to implant, therefore we converted the pseudo-arthritis above his elbow into a working joint. It is a hinge joint, and it functionates well. You will see that he lacks another element, the power to extend his hand or fingers. His musculospiral is out of commission, having been torn apart by the shot, and the ends are separated for a distance of two inches. We are going to transplant his flexor carpi radialis into his extensor carpi radialis, so that when he throws his hand back it will open. If he could open his hand, then he could get a grip. As it is now he cannot do anything with it. He has no power of extension. His lumbricales and interossei muscles produce the little effort at extension he gets now. If he can throw his hand back, he will have a fairly good hand.

Here is one of our prize cases. The little boy,—he was five years old,—had an aplasic fracture of his tibia and fibula. They are the most difficult of all the cases in which to get a union. We have had four or five of these cases. Each patient had been operated on from one to three or four times by various men and always with a failure of union. You can see he had but a little slip of a fibula, not as large as a lead-pencil (Figs. 62, 63, 64, 65). I have not seen this boy since five days after the cast was taken off, but I know that the union is there and it is there to stay.

In this case we used an intramedullary splint in the tibia and a surface graft on the fibula. The peculiar part of it was that the fibula united first. Why? Because it had the aid of that little periosteum above and below that helped to fill in around the graft and hold it fixed to the fibula. There was no union between the ends of the fibular fragments. Osteogenesis progressed through the transplant. That boy has a firm, strong leg now. In place of being a permanent cripple, he will be a useful member of society. That bone will develop to the full size and strength necessary to carry the weight of the body.

Here is another case of the same class (Figs. 66-70). That boy had been operated on four times before we saw him. In one of the operations a Lane plate had been put on. There was a very good approximation of the fragments, but no effort at union.

This accentuates what Mr. Lane has said, that his plate is not for the treatment of cases of non-union. In this case there was no evidence of effort at osteogenesis. *The Lane plate, or any other fixation apparatus, does not stimulate osteogenesis in that congenitally aplasic bone.* That is what I want to bring out.

It is not necessary to have an intramedullary splint. An applied or contacting splint or graft may be used as we did in this case and the one I just cited. We put in the bone transplant in this case in very much the same manner as we did in the other boy. I want you to note the conformation of that limb. It is perfect. There is a splendid union in that leg now.

The size of the transplant does not matter at all. You can transplant fragments as large as the longest shaft from which you are able to take the fragment. In one of our cases we transplanted ten inches of the shaft of the tibia to fill in a gap in the tibia left by an osteomyelitis (Figs. 71 and 72). We had only a button of the internal malleolus left, but we contacted the fragment with that. The upper end was driven into the enlarged medullary canal of the tibia. The operation was done a year ago. That bone has gone on developing and enlarging so that she is able to put her weight on that leg, even bear down on it with her full weight. We recently received a photo from her, walking down the street without cane or crutch. The operation was done in November, 1912.

While we are speaking of fractures I feel I should say a word about a condition that is becoming very common in connection with fractures, that is, the so-called Volkmann's contraction, which is really an ischemic myositis. Here is a patient who came in with his fingers contracted down against his palm. He had had a fracture of his olecranon process and this trouble followed it. He was operated on four weeks ago. We elongated each and every flexor tendon an inch and a half and placed each one in a separate compartment, so as to prevent their subsequent agglutination. We could not find connective tissue enough to cover all the tendons, so we wrapped the remainder in egg-membrane. He moves each tendon separately now. We have had 30 or 40 of these cases.



Fig. 71.—Osteomyelitis of tibia, with destruction of eight inches of the shaft.
Only a small button of bone remained of the internal malleolus.



Fig. 72.—Six months after transplantation of a piece of bone 25 cm. long removed from the crest of the opposite tibia. The upper end of the transplant is in the medullary canal of the tibia shaft, and it is secured with a nail; the lower end is resting in a cavity made in the remnant of the inner malleolus. The new bone is forming along the edges of the transplant.

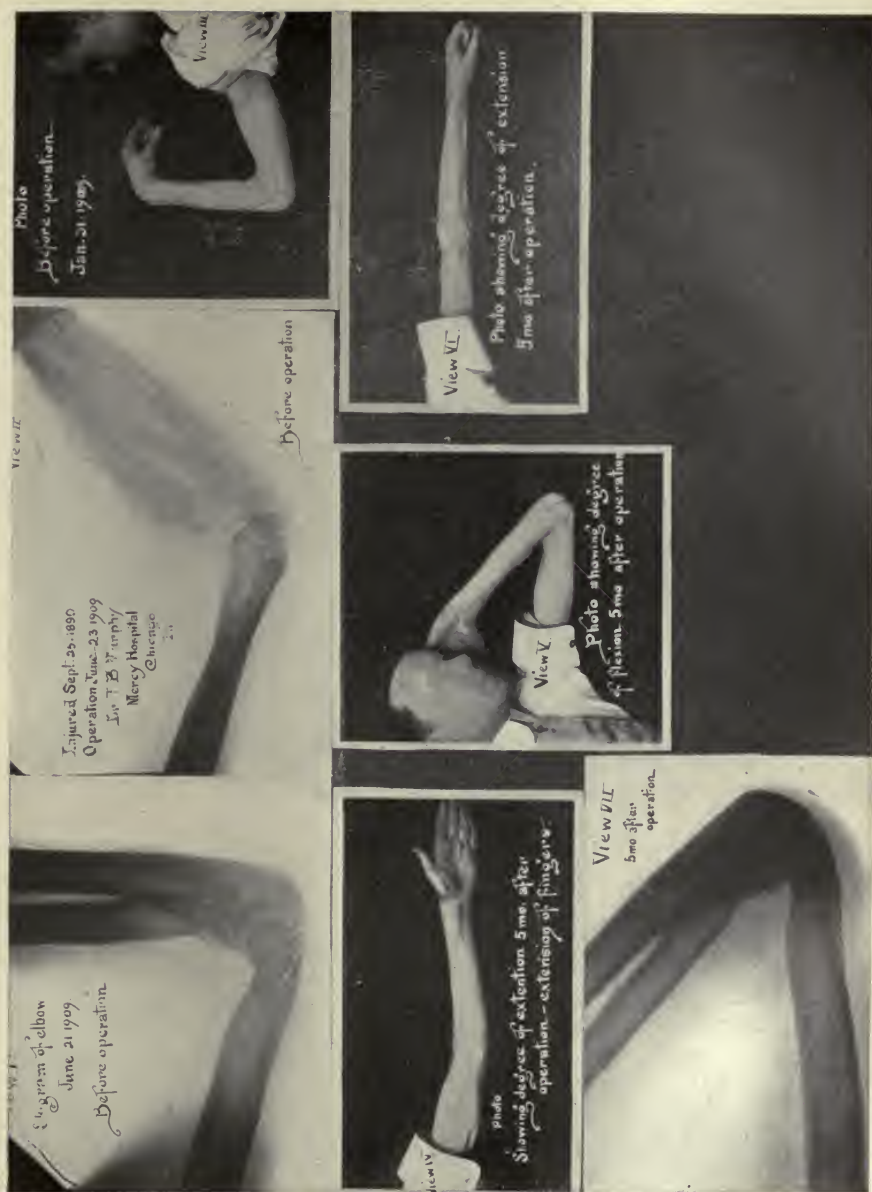


Fig. 73.—Firm extra-articular ankylosis, due to contraction of anterior portion of joint capsule and Volkmann's contraction of flexor tendons of fingers, the latter the result of an ischemic myositis. The capsule was divided, and the flexor tendons were lengthened. The pictures show the position of the elbow and hand before and after operation.



Fig. 74.—Fracture of radius and ulna after first operation. Both bones had been wired and there appears to be some effort at union (compare with later pictures).



Fig. 75.—Lateral view, again showing an apparent union. There is considerable angulation at the seat of fracture, and the later pictures showed absence of union, which was confirmed at the operation done by Dr. Murphy.



Fig. 76.—Fracture of radius and ulna. The ulna fragments had been wired, but there is no union. The radius fragments were plated, and there is apparently a bony union of the fragments (compare with next picture). The relationship of the ulna and



Fig. 77.—Lateral view. At this angle is seen that there is non-union between the radius fragments. The shadow of the radius obscures the ulna.

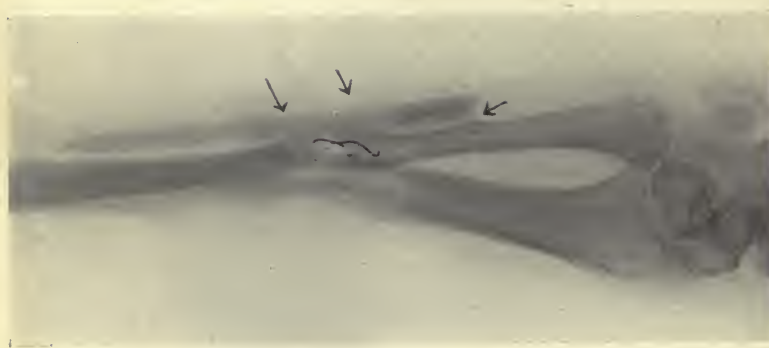


Fig. 78.—Skiagram made about five months after the transplantation (anteroposterior view). The ulna transplant was contacted laterally with the fragments, and osteogenesis is taking place (see arrows). An intramedullary transplant was placed into the radius fragments. Osteogenesis is progressing splendidly, and absorption of the transplant is taking place. The alignment of both bones is good at this angle.



Fig. 79.—The ulna is nearly straight, but there is a forward angulation of the radius. Both bones were solid. Comparing this picture with the first in this series, the formation of new bone in the radius is clearly to be seen.

These cases come and come and continue to come. What are they? They are surgeon's lesions. That is all. Every one is the sequence of a long applied compression. Putting a tight bandage on a limb produces a compression myositis and as soon as the myositis subsides then comes the contraction of the cicatricial tissue and the muscle is shortened. It is never a nerve lesion. *It is never a traumatic lesion. It is always a surgeon's lesion.* That means what? That means that the day of reckoning is coming fast and the thing to do is to get in advance of that day. How can you avoid this condition? You can always tell when it is going to result. When is it going to result? *Always during the first forty-eight hours of the compression.* The myositis is produced by the compression, but the contraction does not take place for several weeks. That means unless you are on guard during the first forty-eight hours, you will have a surgical injury of that forearm. By what? By putting on a bandage, a splint, or a plaster cast that is so tight that it causes edema and swelling of that limb, a destruction of the elements within the muscle-cells, destroying the protoplasm and leaving only the interfibrillar connective tissue, which forms a cicatricial mass, and which has the tendency of a cicatricial mass in any and every position in the body, namely, to contract.

When you take off the dressing at the end of two weeks there is practically no deformity. The patient says when he took off the dressing his arm was all right. *No, it was all wrong! It was all wrong in the first forty-eight hours*—not in the first week, but in the first forty-eight hours.

How are you going to prevent it? If the patient has severe pain in the arm under the splint, bandage, or cast, do not have him wait to call you, *but instruct him, his nurse, his family—anybody—to cut that bandage.* That is all. If you put on a circular cast, *split it before you leave the patient*, and tell the parents or friends to spread it the moment the hand begins to swell and the limb is painful.

Here is another case where the fracture occurred in the lower end of the humerus, just above the elbow (Fig. 73). That took place some ten years before we saw the patient. So perfect

is the union of the humerus that you can scarcely see where it had been fractured, but see the position of his forearm! He could not extend it. There was the position of his hand, as illustrated in the picture. We lengthened all his tendons. First we lengthened his biceps $1\frac{1}{2}$ inches, but the forearm would not come down. Why? Because it was being held there by the contracted anterior capsule of the joint, the result of ten years' fixation. I could not force it down and I felt that if I continued the force I would fracture the humerus before I would succeed in extending the forearm. I divided the capsule by an exposure of the anterior surface of the elbow-joint under the brachialis anticus muscle and the forearm dropped down. That is the best way. Divide the capsule; do not endeavor to fracture it. Four weeks later we lengthened all the flexor tendons of the forearm. That man can move all his fingers separately, and not only that, but he plays the piano very well. We elongated each tendon from $1\frac{1}{2}$ to $2\frac{1}{2}$ inches. That shows what can be done in these cases.

Here is another case of non-union—an illustration of the same condition of non-union we had this morning, but a little lower down in the forearm. It was a fracture of both bones of the forearm. The patient was operated on four months ago. We are keeping the arm perfectly still, because it is a case of transplantation. You see the position of the arm is good. Pronation and supination are restored, but she continues to carry that arm perfectly still until we know to a certainty that the regeneration is perfectly secure (Figs. 74-79).

Here is another case of Volkmann's contracture. The patient is a doctor who was my intern some years ago. He was operated on ten years ago. He had a fracture of his forearm with a contraction myositis. We lengthened his flexor tendons in the usual manner. He has a full and complete restoration of function in his hand, and that is ten years after the operation.

Let the record show that there was no union between the ulna; that there was a distinct cicatricial capsule around the bone, which was removed; that a piece of bone was taken from the tibia and inserted between the two ends, after they were prepared in the usual way.

LUXATION OF THE PATELLA AND FRACTURE OF THE INTERNAL SEMILUNAR CARTILAGE; DESCRIPTION OF DR. MURPHY'S OPERATION FOR LUXATION OF THE PATELLA

HISTORY

PATIENT, female, aged twenty-six, entered the hospital November 7, 1913, because of weakness of, and constant dull pain in, both knees, and an occasional locking of these joints.

Present trouble was first noticed four years ago, when the patient turned quickly and fell to the ground because of severe pain in the right knee and a locking of the joint. She was able to get up on her feet after a few minutes, after which the sharp pain subsided. She could walk, but the knee was somewhat painful and stiff for several days, causing her to limp. There was no swelling. From this time on she had no severe trouble with the knee, but always had a fear, when walking, that the knee would lock. There was a more or less constant dull pain in the right knee, which seemed to be exaggerated during damp weather. About three years ago, while reaching up with her arms, she developed a locking of one of her knees, but does not remember which one. The pain was not so severe this time, and after a few minutes it practically subsided, and she was able to walk. Since that time she had another locking of her knee-joint, but does not remember which joint or just when it occurred. Her knees have been weak ever since. The feeling on walking is as though they would give way, and she constantly fears a locking of them. There has been a constant dull pain in the knees nearly all the time, and during the last three or four months they have become very weak. The pain is worse after walking, and she has some difficulty climbing stairs. No other joints are affected. There is no history of infection or trauma. Family and personal history otherwise negative.

COMMENTS AND OPERATION

DR. MURPHY (November 10, 1913): No, no! that is not the true story. She did not turn quickly and fall. She turned quickly, had severe pain in the knee, and because of the pain she fell. What an entirely different story that is! She did not fall and injure her knee. She injured her knee and fell. I see practically all these cases in my office first, so that I know the story. She told me that she made a sudden turn, had severe pain in her knee, and fell. When she got up she could not straighten her knee. She hobbled around for a while, and finally she got the leg straight. What a different story! and the story is so important in making the differential diagnosis. What is the matter with her?

INTERN: She has a dislocation of the semilunar cartilage.

DR. MURPHY: What makes you think so?

INTERN: The symptoms.

DR. MURPHY: What symptoms?

INTERN: The locking in the knee-joint.

DR. MURPHY: What else?

INTERN: She had pain in the knee which was followed by a locking of the joint.

DR. MURPHY: No, this is the way it occurs. They make a sudden turn, the semilunar cartilage slips out of position and locks the knee in that position. After a time the cartilage readjusts itself, and the joint comes back into position, but the pain and the locking are synchronous when it is a case of the classic type. This girl did not fall first. The pain was so severe that she went down too far on account of the pain. She did not go too far and then have the pain—just the reverse.

Two conditions are combined in this case, and it is very difficult to differentiate them from the patient's history. They are an external luxation of the patella and the luxation of the semilunar cartilage. Unless one is extremely careful, he will mistake one for the other. Here we have both conditions combined, and this is the first case of the kind we have had.

I remember one of my early cases of luxation of the patella.

The patient came to the office and related a typical history for a luxation of the semilunar cartilage. She said she had the trouble for four years and that she had been all over the country trying to find out what it was. Making the examination in the regular way and putting the patella through the usual range of motion, I was struck with the fact that the range of motion was greater than normal, and particularly the degree of external displacement. I had her close to the edge of the straight table on which she was being examined, and kept moving the leg a little nearer to the edge with my fingers, manipulating the patella until I had it displaced outward onto the external condyloid ridge, which is flat, an inch or an inch and a half above the articular surface. Then I suddenly dropped the leg off the table, and she screamed, "There it is! there it is!" It was not a semilunar cartilage displacement at all. It was an external luxation of the patella. She could straighten the leg every time. One must be very mindful of luxation of the patella as well as of luxation of the semilunar cartilage.

Two English surgeons gave us most of our information on the displacements of the semilunar cartilage. Mr. Owen first described them, and then subsequently Mr. Morrison, who has had such an enormous number of these cases, worked out the treatment. Three years ago he told me that he had up to that time treated over 500 cases of luxation of the internal semilunar cartilage. He is located in the Newcastle coal-mining districts. The miners work in a stooping position, and luxation of the semilunar cartilages—usually the internal—occurs very commonly in this region. It is the most common cause of articular trouble. The reason we do not diagnose the condition more often is because many of these cases are a fraying-out or loosening of the internal semilunar cartilage, not a fracture, and the symptoms are those of a continuous pinching of the small filaments of cartilage, often not as large as a No. 3 silk thread, that stretches across the surface of the joint and gives the patient an enormous amount of trouble. It is the source of a constant irritation and inconvenience and even hydrops.

The removal of the cartilage is the only means of effecting a

cure in these cases. These luxations occur most commonly in athletes, and next most commonly in those who work in the coal-mines, as in Mr. Morrison's cases. In the management of those cases at Newcastle the men are sent to the hospital at once, and the cartilage is taken out. The number of infections that have occurred in Mr. Morrison's cases has been reduced to a minimum, so that the possibilities of danger, I would say, would be about those of an interval appendicitis operation.

Removing the semilunar cartilage when the joint is opened is not a difficult proposition. In this case we have to do something to prevent a recurrence of the external luxation of the patella. Some years ago we worked out a method for controlling that. In the article we wrote in 1912 on the surgery of the bones and joints, published in *The Journal of the American Medical Association*, we laid particular stress on the fact that the fibrous capsule of the joint is inelastic. You can fracture it, but you cannot stretch it. We take advantage of that fact in the treatment of this luxation of the patella. After having split the capsule we make an imbrication of the internal flap of the fibrous capsule of the joint—put one flap under the other, a typical overlapping or imbrication. That effectively prevents the patella from being luxated again onto the external condyloid ridge. Both these operations will be carried out in this case through the same incision.

The patient is ready for operation. She lies flat on her back on the table with the legs hanging over the end of the table and flexed at right angles to her body, so as to give us good access to the internal semilunar cartilage. We always sterilize the skin with a half-strength (3.5 per cent.) tincture of iodine, but we neutralize the iodine with alcohol before making the incision, because we seal the wound with collodion at the completion of the operation. If you put collodion over the iodine, in 10 per cent. of the cases a dermatitis will be produced. That dermatitis is serious from the standpoint of infection, and for that reason we neutralize the iodine in all operations in the neighborhood of a joint. The iodine had accomplished its purpose. It has filled the epithelial cells, and the alcohol removes the excess.

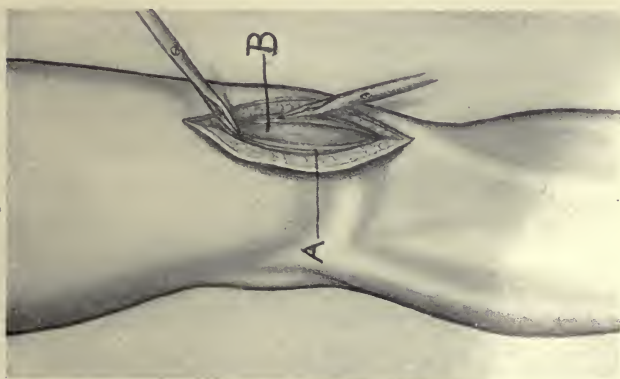


Fig. 80.—Luxation of patella. The outer fibrous layer of the capsule (A) has been divided and undermined preparatory to the imbrication. The inner layer (B) is intact; the joint cavity has not been opened.

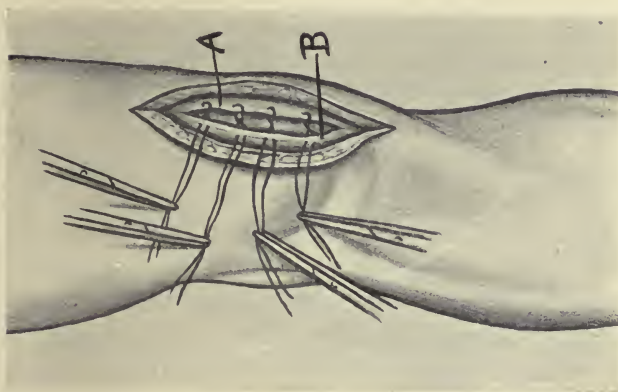


Fig. 81.—The sutures have been passed through the edge of the internal flap, A, and back from the edge of the external flap, B, so that the external flap will overlap the internal when the sutures are tied.

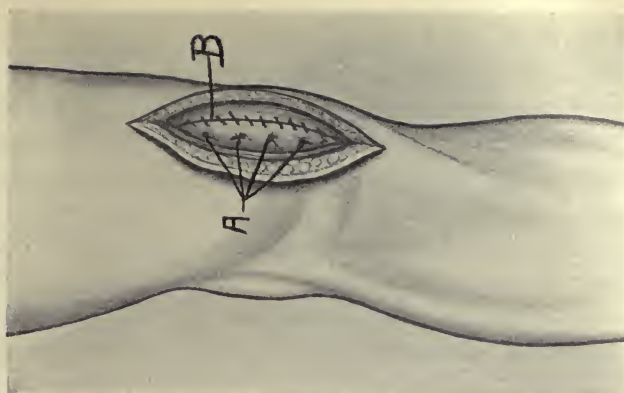


Fig. 82.—The sutures have been tied; the knots (A) show where the edge of the internal flap has been drawn under the external; the sutures B tack the edge of the overlapping portion of the external flap to the outer surface of the internal flap.

I make the incision along the inner margin of the ligamentum patellæ, about $2\frac{1}{2}$ inches in length, and passing over the internal tuberosity of the tibia so as to give me a good view of the joint. I cut down on the fibrous capsule very carefully and my assistant and I grasp it with tissue forceps, incising it between the forceps just as we do when we open the peritoneal cavity. The inner layer of the capsule, the synovial membrane and its subendothelial lymphatics and blood-vessels, is the zone in which the hematogenous infections occur in this neighborhood. Now we free the fibrous layer of the capsule for $\frac{3}{4}$ inch from the sub-fibrous layer of the synovial membrane, so as to have it ready for the imbrication or overlapping when we proceed to close the joint. We cut through the synovial membrane very carefully so as not to injure or abrade the mesothelial cells which line it. You will note that we do not touch the field of operation with anything except sterile instruments. Not even the gloved finger touches the wound. The skin is never touched, because it is the source of great danger so far as infection is concerned. If I injure the synovial membrane, ankylosis may occur.

Now the knee is open. There is the synovial fluid. I can see the interior of the joint. There is a fracture of the internal semilunar cartilage. The ends are sticking out into the wound. I grasp one end with forceps and cautiously free the cartilage with dissecting scissors. Now I have freed the cartilage and it is easily removed from the joint.

The edges of the cartilage where it has been fractured are thin and shining. The synovial membrane is not materially thickened. It is, however, more resistant to infection than it would be if it had not been the site of a long-continued inflammation. You will note how careful we are about touching these surfaces. We keep the field sterile, and we do not traumatize these tissues. The bleeding has all ceased. There is rarely much hemorrhage in these cases.

The incision in the synovial membrane is closed with a continuous looped chromicized catgut suture, with an eversion of the edges of the wound so as to prevent the occurrence of adhesions subsequently.

Now we are ready for the imbrication of the external or

fibrous layer of the capsule for the cure of the luxation of the patella. We will do that on the same basis as the Andrews hernia imbrication operation is done. We will pass three or four sutures of plain catgut through the edge of the external lip of the incision, and draw the flap under the internal flap, as follows: The needle is first passed through the internal flap, about one-half inch back from the edge; then it picks up from underneath the edge of the external flap, is carried back under the internal flap, and emerges within one-eighth inch from the starting-point. When this suture is tied, the edge of the external flap is drawn under the internal flap. That makes the imbrication. We insert three additional similar sutures to insure a good approximation. The internal capsule is, therefore, narrowed well up into the vastus internus or made tighter really so that the patella cannot slip again from its bed.

Now the free edge of the internal flap, which is on the outside of the external flap, is secured to the latter by means of three or four tacking sutures of catgut, and the imbrication is complete. We will make that fixation of the patella doubly sure by fixing the inner edge of the quadriceps tendon to the aponeurosis of the vastus internus. Now that patella cannot luxate again, as the first step in the external luxation of the patella is when it starts to slide on the outer side of the supracondyloid ridge high up, and continues on the outer side of the ridge until the luxation is complete. It is, therefore, important to shorten the vastus internus high up. We believe that this operation is original with us. It is the overlapping flap that gives it the strength. It just cannot get away. If I did not have to remove the semilunar cartilage here I would not have had to open the true synovial membrane of the joint at all. That is the value of that operation. There is no constrictor on this leg, and yet there is little bleeding, and this we check as we go along with hemostats. You rarely ever have to put on a ligature. If the bleeding is severe, we just put on a Blunk hemostat.

All that remains to be done now is to close the skin wound with horsehair, and seal it with collodion gauze. We have been exceedingly careful about our asepsis. Nothing except sterile instruments came in contact with any part of this wound. Not

even the catgut was handled, except when I tied the sutures and that portion of the gut was outside of the knot and was cut away. Remember that the capsule of this joint consists of two layers—one the external fibrous layer, which is inelastic, practically non-vascular, and never the seat of a primary metastatic infection, and an inner layer, the synovial membrane. The synovial membrane is a “serious” membrane, from a surgical standpoint, as well as a “serous” membrane. Every metastatic infection into the joint coming through the blood lodges in the vascular and lymphatic layer of that membrane, never on its surface, except in the streptococcic type of infection. Is that clear? In other words, the so-called infections in the joint are not infections in the cavity of the joint, but infections of the vascular tissue outside of the mesothelial layer of cells lining the inner or joint surface of the synovial membrane. You can easily dissect that free from the fibrous capsule without opening it. However, we are careful not to infect any part of this wound. A plain dressing will be put on this knee. The stitches will be removed in about ten days.

We always put a Buck's extension on the lower leg in these cases, with a 15-pound weight attached. After two weeks the patient is allowed to be up on crutches, gradually putting a little weight on the leg. When the joint is strong,—say, after six or seven weeks,—the patient discards the crutches and walks without any support.

Let the record show that there was a fracture and fraying of the edge of the internal semilunar cartilage; that this was removed and that we made an overlapping of the fibrous capsule in the regular way.

Let me show you an interesting case, the first of its kind I ever saw. This boy has a congenital external luxation of both patellæ. He can walk as well as any one can, and he can run as well as any other boy, but he cannot stop running without falling down, and if he stubs his toe he falls. Flex the leg and there is no patella to be seen. The skiagram does not show a patella. I had to look this boy over quite a while before I finally located the cause of his trouble, a congenital external luxation of

his patellæ. It was resting on the outer aspect of the external condyle when the leg was flexed. It is a congenital luxation.

We operated on the left leg, leaving the other leg to be operated on later. He has a complete primary union of all the parts, and there is the patella in the midline. We had to make an extensive dissection to free that patella and bring it into its normal position. We made a bed for it with a curved chisel in the usual location on the femur because there was no intercondyloid notch. We used the curved chisel and made a good bed for the patella.

The knee is now entirely normal. He can flex it without dislocating his patella. It remains in its normal position. The reason he could not stop running without falling down was that his quadriceps was acting as a flexor instead of an extensor, pulling his leg back onto his thigh so that he had to fall. The boy can now run and stop. He flexes the knee to better than right angles, and the patella remains in its new bed. He has full extension.

After freeing it from the outer external surface of the external condyle and extending that freeing over the vastus externus three inches above the joint and below and downward to a level with the tubercle of the tibia, we were then able to bring the patella to the midline. We then divided the fibrous capsule on the inner side of the patella half an inch from its edge, and extended that division three inches upward. We next elevated the internal flap over the patella and attached it to the external anterior surface of the ligamentum patellæ, the patella, and the stump of the vastus externus, so that it really made an overlapping flap for the entire length of the patellar ligament and the quadriceps attachment of the patella, and gave us a secure fixation.

Here is another case of external luxation of the patella. She had practically the same symptoms as the patient we operated on today, although she did not have a fractured semilunar cartilage. The semilunar cartilage was not disturbed, differing from this case today. Twenty days have elapsed since we operated on her. She is commencing to go about on her crutches. That joint was not opened at all.

We have had about seven cases of luxation of the patella up to the present time, and the results have been good in all.

LAMINECTOMY FOR TRAUMATIC COMPRES- SION OF THE SPINAL CORD

HISTORY

PATIENT, male, aged twenty-four, comes to the hospital with the following history:

On August 27, 1908, at 8.30 P. M., while driving a team of horses, it ran away. The wagon turned turtle, with the patient beneath. He was stunned for a second, but never really lost consciousness. From the ecchymosis that followed it was thought that a friend, who was with him, had fallen so as to strike the patient with both knees in the lower dorsal region and on either side of the spine. There was an immediate paralysis of the legs when the shoes were removed, fifteen minutes later. Five days afterward, September 1st, an operation was performed, at which time there was, as immediately after the accident, no sensation, no motion, no sensation of heat or cold—thus, a total paralysis of both limbs and the lower part of the abdomen, up to one finger's breadth above the umbilicus. The patient states that the injury was found about the eleventh or twelfth dorsal. A piece of bone had been driven into the cord at the twelfth dorsal and this was removed.

He has noticed that since the operation there has been at times a twitching in the legs, but no voluntary motion. A catheter has been used more or less for about a year. Bed-sores come and go in the left side.

Examination.—It appears as if all below the tenth dorsal vertebra on the left side and the ninth on the right side was paralyzed. A large arched deformity with convexity posteriorly is made by the spine at the twelfth dorsal to the first lumbar. Lately Babinski, ankle clonus, and knee-jerks were found present. Priapism exists most every day about 4 to 5 A. M.

EXAMINATION BY DR. CHARLES LOUIS MIX, DECEMBER 6, 1913

REFLEXES:

Cutaneous: Plantar, present and reduced.
Abdominal and epigastric absent.

Muscular: Cremasterics absent.

Tendinous: Right knee-jerk present; left absent.
Ankle-jerks absent.

Abdominal: Right Babinski slight; left Babinski marked.

SENSATION: Abdominal—upper level at ninth rib or ninth interspace.

Zone of hyperesthesia, three inches wide, immediately below.

Anesthesia below the hyperesthesia zone.

Testing of sensation in hyperesthetic zone causes tingling sensation below, particularly in the right leg.

MOTION:

Voluntary: Abdominal muscles, especially rectus, can be used; none below.

Involuntary: Fibrillary movements and spasms of muscle portions mostly in the right, but also in the left leg and thigh.

CONTRACTURES: Never present.

ATROPHY: Even, of the whole lower extremities; perhaps more in the right.

No selective atrophies.

BLADDER: No sphincteric control. Dribbling for four and one-half years.

COMMENTS AND OPERATION]

DR. MURPHY (Operation, December 8, 1913): That is a typical history, as you see, in some particulars. The first is that, from the story, there was apparently an immediate paralysis. When the paralysis is immediate, instantaneous, and annular, the rule is that the cord is divided. That is difficult to determine unless a doctor is present to observe and carefully note what has happened. The edema which then sets in rapidly following trauma causes a suspension of transmission of motion and sensation through axons that are traumatized, *though not actually divided*. Now, then, what has happened? When the physician first examined this case there was a paralysis of motion and sensation,



Fig. 83.—Laminectomy. Skiagram, lateral view, showing the angulation of the spine at the first lumbar vertebra and the compression of the anterior portion of the body of this vertebra.



Fig. 84.—Laminectomy. Skiagram, anteroposterior view, showing a perfect alignment of the spine. The first lumbar and twelfth dorsal vertebrae throw a much darker shadow because the angulation of the spine is at these vertebrae, and, therefore, they were nearer the plate.

but that was a considerable time after the injury. At the operation a spiculum of bone was removed from the spinal cord and the flaps of the dura restored. The spinous processes were taken out and left out. At present he has, after five years, a return of the reflexes, a return of the Babinski, a return of priapism, which means what? That he has within that cord a number of living motor axons, otherwise these things could not take place.

He was injured in the caudal zone—the twelfth dorsal, and the lower part of the eleventh. The upper part of the eleventh is normal. In looking over the patient it appears to us that there is a compression, either at the line of the removal of the spinous processes or perhaps lower down. In analyzing this case we decided that the cord must be exposed and freed from bony or cicatricial compression; free the zone below it, also above, and then we may be able to say what will be the final outcome of this case. He has had a decubitus; that has practically healed, except one little zone. One never likes to operate when these are present, because there is a certain amount of danger, first, of surface, and, second, of hematogenous infection.

In the skiagram you will see there is a marked deformity. That is a splendid skiagram. You see the relation of the heart. The lung between the spinal column and the heart; the heart between the two arches made by the diaphragm and the liver. You see the cavity in between. Then again the lung between the heart and the spinal column. Then again the lung between the heart and the chest-wall in front. I think that is probably the best transverse picture of the chest that I have ever seen.

This will be a slow operation. We must go in and locate the spinous process above and below the ones that were removed in the previous operation. Remember, it is much more difficult to reoperate where a laminectomy has been performed, because the surgical landmarks are destroyed. Just below the point of the injury he has a very marked lordosis. His greatest callus formation, a soft one and a callus that obstructs the x-ray, is just below the twelfth rib. You feel it there in that position. Let me show you the darkest line, giving you the impression of

a solid mass. It is not a bony mass, of course—it is merely soft tissue, cicatricial formation that grows around the spine when it is fractured and helps in fixing the spine when the normal bony support of the bodies is gone. What about the urine?

INTERN: No albumin, no casts, occasional white cells, and triple phosphates.

DR. MURPHY: He was catheterized in the beginning. How long?

PATIENT'S PHYSICIAN: Off and on for a year.

DR. MURPHY: That is an exceptional result to find at the end of a year, if that report of the urine is correct. While we have some reasons to believe it is, we have many reasons for believing it is not. However, if that report is correct, that is a very exceptional condition to find at the end of five years. The causes of death in the order of frequency following fracture of the spine are, first, from the decubitus and sepsis from the bed-sores. Second: the infection of the urinary tract from the catheterization, with an ascending infection into the kidney which causes a pyelonephritis—the nephritis or surgical kidney from which these patients die. More of these patients die as a sequence of surgical kidney than from any other one cause except decubitus.

[Showing patient.] You can see the lordosis I spoke of 4 inches below the injury, which is going to make it difficult to handle. Nothing in his spine to indicate there was a fracture down where the anterior curvature is, but it seems, from the position of the abdomen, etc., that that has developed as a compensating lordosis.

[Incision.] There is some venous bleeding here, and we will just pack that a little and then we will commence to free this process below. There is the greatest deformity. There is the greatest angulation. The top of the process, which was not taken off in the previous operation, was turned so that it does not appear to be quite normal. Here is where there appeared to be the greatest luxation—at this point, the first lumbar body. Notice the prominence of the first transverse process. See how nature has filled in the gap between the ends of the laminae! This shows the lamina above where the twelfth was removed.

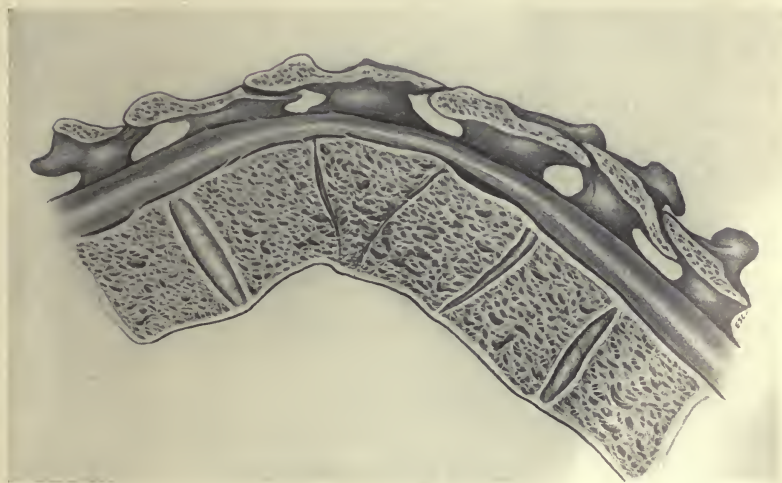


Fig. 85.—Laminectomy. Longitudinal section of spinal column, showing the compression of the anterior portion of the body of the first lumbar vertebra, causing an angulation of the spine and compression of the cord.



Fig. 86.—Laminectomy. The transverse processes and laminae have been removed, and the cord is exposed. The dura has not been opened.

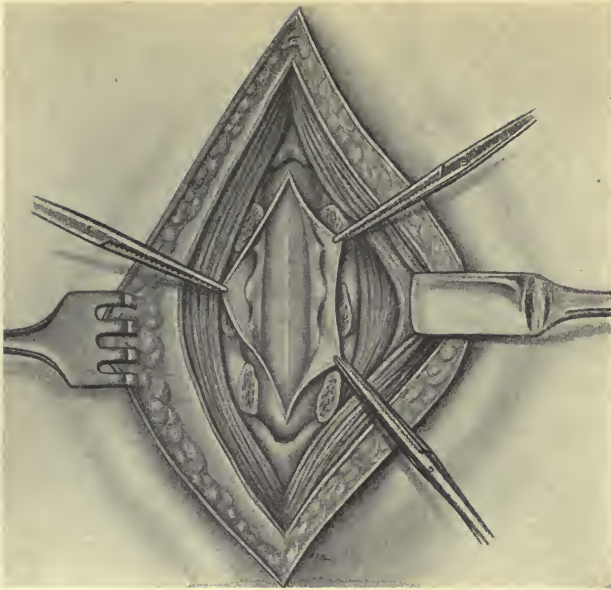


Fig. 87.—Laminectomy. The dura has been opened, and is drawn to both sides to expose the cord, which is flattened out by the cicatricial tissue running across it. (This is a schematic drawing and does not show the scar tissue.)

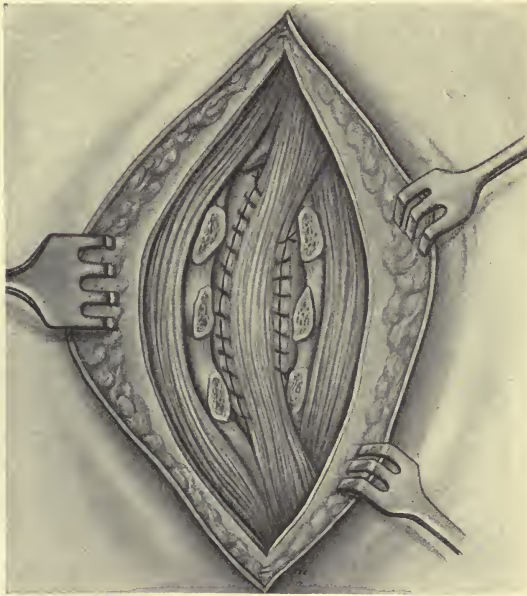


Fig. 88.—Laminectomy. A strip of the erector spinae muscle has been drawn over and sutured into the gap in the dura to prevent subsequent pressure on the cord by cicatricial tissue, a procedure which is original with Dr. Murphy.

Here there is, you see, something pressing backward against the spinal cord at this point. There you see the indentation in the cord. There is where it comes through the elevation and where we apparently have our trouble.

The best instrument for removal of the osseous framework of the cord is the small ronguer forceps with a flat lower blade which is not fenestrated. One can go under the bony structures and can always pick them up without danger of injuring the cord. With each additional pressure of the forceps we pull off the periosteum from beneath. There is where the cord is compressed. That is where it is injured. See the difference in these conditions! That is just beautiful! There is the edema, one-half an inch below the constricted point, just as we estimated it from the x-ray and clinical history. Now we have the tissues entirely freed of the bony overstructures. There is the normal cord below—the cauda.

Now I am splitting the dura from its free position below the injury through the cicatricial tissue to a free and normal dura above the injury. The cerebrospinal fluid is escaping. Here is cicatricial tissue on and in the cord, the result of an old hemorrhage. Now we will determine with the faradic current whether the axons are alive below the cicatricial tissue. It gives a positive response of the muscles of the lower extremity. Look! You rarely have an opportunity to see this demonstrated: the axons are alive below the cicatricial tissue in the cauda; they must, therefore, be alive through the cicatricial tissue, as these cell bodies are situated above the cicatricial tissue in the conus or in the cord above; but suspension of transmission in the living axons is effected through the compression of the cicatricial tissue of the dura and the compression produced by its contraction. The axons are living through the cicatricial tissue, else we could not have a faradic response below it. Before freeing the cicatricial compression by dividing the dura and subdural scar tissue parallel to the long axis of the axons, let us apply the faradic current proximal to the scar tissue and see what the result will be. Before dividing the scar tissue I will go above it and apply the

current. Now we fail to get transmission of impulses through the cicatricial and compressed zone.

Let the record show that above the point of compression there is a twitching of the lower abdominal muscles over a zone of one-half inch faradic application. This point is where the greatest trauma was with the angulation and scar compression from the lacerated dura. I have split the dura for a distance of 3 inches here, parallel to the long axis of the cord, so as to make a muscle transplant into the cicatricial zone of the dura and spread the ends of the scar and prevent the transverse compression of the scar tissue. Again applying the faradic current below the scar on the lower axons, going in from the posterior side, both legs move. Now let me go in from the other side and get the anterior muscles. Both legs also move.

Let the record show that the flexor and extensor axons of the foot, leg, and thigh are all living.

That agrees with our interpretation of the case before operation, namely, that the axons of the cord were living, but that there was a suspension of transmission; *i. e.*, that they had not been divided, but were compressed by cicatricial tissue.

I am freeing the cord on its side from the scar tissue, so as to make a transplant of fascia and muscle between the cut edges of the dura scar tissue, and get all the pressure off the cord, so that it can expand indefinitely in a backward direction. Now I am up to a normal pulsating cord above the scar tissue. The cessation of pulsation extends over a zone of $1\frac{1}{2}$ inches. The lowest point of that is at the margin of the second lumbar, and the highest point is at the lowest margin of the twelfth dorsal. Now that the cord is free we will test the faradic conductivity of the axons from above through the scar tissue. It reacts. That is the first transmission we have had from above the scar since we began this operation.

Let the record show that after freeing the cicatricial tissue that held the cord transversely compressed, we were able to get transmission through the scar zone for the entire length; we have, first, splendid transmission through the axons to all the muscles, with the faradic current applied below the point of

compression; second, that during the continuation of the compression of the cord by the cicatricial tissue there was *no* transmission of faradic current when applied from above; third, that after dividing the cicatricial tissue and the dura, thus freeing the compression of the cord, we had free transmission from above to all the muscles through the formerly compressed zone of the cord. This shows positively that the axons were alive.

Get that very clear, because it is very important. That completes the proposition. Let us prepare to repair it. Now, then, I must do what? If I sew the split dura back again he will have exactly the same transverse compression of the axons that he had before. We must fill in the gap in the dura with something. We will use a plan we have followed before taking this erector spinæ muscle with its smooth fascia from the side, and throw the muscle forward in between the edges of the dura, turning it over with its smooth fascia toward the cord and suturing it to the edges of the dura so as to get a $\frac{3}{4}$ -inch expansion of the lumen of the dural canal after the implantation is made. (See Fig. 88.) This insures a permanent release of the cord from transverse dural compression. Now, you see, I have freed that flap. This is fibrous tissue; it lives by osmosis rather than by vascularization. That is why you can use it in this position. This is a new procedure as far as we are able to find out, and was original with us, we believe, though it is dangerous to claim originality in anything since the excavations in Asia Minor have disillusioned us on so many supposed modern originalities. Now, you can see how this is put in. There is the divided dura. I am taking this fascia and uniting it, suturing it between the edges of the dura. There is the top of the dura. Here is my flap on the right side. Note how I have that attached to the dura. That is cicatricial tissue—the upper part of the original scar. There is the margin of the dura that I am uniting the interposed flap to all the way.

Here is the other side of the dura, way out here. See how I interpose that flap of fascia and fatty tissue which was entirely outside the dura in between the divided margin of the dura. There is the margin of the dura. I am holding it elevated.

Let the record show that the cerebrospinal fluid flowed out below the point of trauma, but there was no communication across the scar or cicatricial tissue zone with the fluid above.

Note.—It will be many months before the results of this operation can be demonstrated. This case shows, even after five years, that the axons were living through a cicatricial mass, but on account of compression, transmission of impulse was suspended. We have demonstrated this same type of suspension of axonal transmission in the musculospiral compression in fractures of the humerus where the nerve was not divided.

REMOVAL OF ENLARGED AND DILATED STUMP OF GALL-BLADDER FOLLOWING A PREVIOUS OPERATION, WITH SECOND- ARY PERFORATION OF ITS WALL BY THREE CALCULI

HISTORY

PATIENT, male, aged forty-five, entered the hospital November 16, 1913, because of a constant dull pain in the right hypochondriac region and general soreness and discomfort in the right side of the abdomen.

In 1897 he had night-sweats, pain in the right upper part of his chest, cough, and expectoration of bright red blood. He was told that he had pulmonary tuberculosis, and went to Colorado for fifteen months, submitted to anti-tuberculosis treatment, and recovered. In 1905 he had a similar attack of illness and again he went west, but only for three months. In July, 1906, he was coughing considerably, and tubercle bacilli were found in his sputum. He again went west, this time for thirteen months, and since then he has had no more attacks.

October 24, 1907, the patient was operated on for gall-stones. Eleven large and 11 small stones were removed, and the gall-bladder was drained for ten days. The tube was then removed, and two and one-half weeks later he left the hospital with the wound completely healed. For ten years previous to this time he had had intermittent attacks of pain in the right hypochondriac region. Occasionally these attacks were accompanied by chills and they were always followed by vomiting of bile and by jaundice. The urine always was dark in color and the stools clay-colored during these attacks. After leaving the hospital he felt perfectly well until three years ago (1910), when he had a chill, which was followed by pain in the right hypochondrium. He

does not remember whether he vomited or became jaundiced. He remained in bed for two weeks, however.

Some time in the fall of 1911 he again had a severe pain in the right hypochondriac region, but he did not vomit nor did he become jaundiced. On palpating his abdomen he discovered a tumor in the left groin. It was about the size of an egg, rather firm, and slightly tender to touch. The tumor was removed and examined for tuberculosis. It was not a tuberculoma. He remained in the hospital ten days. Since that time he has felt fairly well, except for occasional feelings of distress in the epigastric region, which had no relation to meals. He did not vomit or become jaundiced.

In October, 1912, he had a severe attack of pain in the right hypochondriac region and vomited excessively. The vomitus contained bile. He was also jaundiced. This attack continued for several days. Then he remained quite well until three months ago (August), when he noticed that on awakening in the morning he had a feeling of soreness in the region of the gall-bladder and also in the appendiceal region. He did not vomit, but he was jaundiced off and on until two weeks ago, when the icterus disappeared completely. At this time he went to the hospital. He was put to bed, given a gallon of water to drink daily, and put on a liquid diet. He remained there until last night, when he came here. Since the operation on his gall-bladder his stools have been clay colored off and on, the urine at the same time being dark, but never containing blood. His bowels are regular; no diarrhea; appetite good. Leukocyte count, 12,800.

COMMENTS AND OPERATION

DR. MURPHY (November 17, 1913): The interesting part of the story is that he had chills and fever associated with jaundice. We do not know definitely, however, whether it was a real jaundice or whether it was just a tinging of the skin that many people call a jaundice, but which is not to be interpreted as a calculus obstructive jaundice at all. If he was jaundiced, I would say that without a doubt it was a stone in the common duct. An individual may have chills and fever or even pain,

nausea, and vomiting for months and years without ever having jaundice. We have a patient in the hospital now who had been having chills and fever of a very severe type for two years and finally got some pain in the right hypochondrium. The case was diagnosed as "ague" by the medical attendant.

It was a septic gall-bladder. The cycle of chills and fever resembles exactly the chills and fever of ague, except when there is a stone in the common duct. Then there may be associated with the chills and fever a jaundice or a colic if the stone is moving. Without the colic and jaundice it is difficult to differentiate the chills and fever of "ague" from the chills and fever accompanying a septic cholangitis, except by the leukocytosis, which always tells the story. A man who has chills and fever from a septic cholangitis, with or without the impaction of a stone in the common duct, always has a leukocytosis.

These cases were being treated for years for "chills and fever." I remember one man who was treated for the "ague" for eleven years. He traveled all over the world to get out of the ague zone. Finally he went to California, where he had the chills and fever first every seven days, then every second day, and then three or four times a day. He would have a chill and a temperature of 104° F., and then the temperature would drop right down to normal. Many years ago I called attention to that sudden drop of the temperature. I termed it the angulation temperature of septic cholangitis (Sajous' Medicine). It is a striking contrast to the temperature curve of sepsis elsewhere in the body. It resembles the chills and fever that accompany a portal thrombosis with infection. The temperature is high for a couple of hours in the day, then drops to normal. These chills and fever may occur when the gall-stone is in the common duct or when it is impacted in the cystic duct, or when the infection is severe in the gall-bladder. When the stone is in the cystic duct, you have the chills and fever without jaundice. When it goes into the common duct, you have it associated with jaundice. For years this was known as a malignant malarial jaundice. This was first described by Trousseau and later by Dr. Wm. E. Quine, of this city. He cited four postmortems on cases in which the stone

remained in the common duct. The patients had had the typical chills and fever that occur in malaria.

We will open the abdomen by going through the aponeurosis of the right rectus muscle.

Secondary operations in any portion of the abdomen are very disagreeable, and particularly so in the upper abdomen. There is a good union of all the layers of the abdominal wall from the previous operation.

I have succeeded in getting into the peritoneal cavity below the point of adhesions. I will endeavor to expose the line of fixation of the gall-bladder. All adherent on the outer side and on the inner side. Now I am on the lower margin of the liver, and I am in a zone free from adhesions. The liver is bound firmly to the anterior abdominal wall on the outer side. I will get the omentum free. I am freeing it. I feel a mass of stones on the under surface of the liver or in the gall-bladder, I do not know which, but they appear to be outside of the gall-bladder and on the surface of the liver. I am freeing this omentum from the liver, otherwise I will be unable to get at the gall-bladder.

I can now raise the edge of the liver. See that gall-bladder! It is about two inches wide and an inch and a half long. It is firmly adherent to the liver throughout its entire extent. There are the stones coming out. See them, just ulcerated through the gall-bladder!

That looks like the fundus of the gall-bladder with the stones ulcerating through. One stone has perforated and is in an encapsulated pocket outside of the gall-bladder; a second is just coming through, and a third is almost through. It is just underneath the serosa; nothing but a little fibrous tissue covers it. There is the cystic duct. It is angulated and contracted about half an inch from the gall-bladder. There is every evidence here of a former septic cholecystitis. There is the hepatic artery.

I will now examine the pancreas. Let the record show that the head of the pancreas is somewhat thickened—not great enough to say that it is a malignant condition. I believe it is not malignant. Let the record further show that the common

duct is somewhat thickened; that there is no stone in it, nor is there a stone in the cystic duct.

I will remove the gall-bladder. There is a great deal of cicatricial tissue here and we must proceed carefully. I will divide this cystic duct and try and get this gall-bladder out from behind. We will divide the duct between two clamps and tie each end separately with a No. 4 catgut ligature. The proximal stump of the duct is then touched with iodine on a cotton applicator. There is no bile in the gall-bladder. It was flattened out like a fan, and firmly adherent to the under surface of the liver. We will tie the vessels separately. I amputated the gall-bladder just at the junction of the gall-bladder with the cystic duct. I will fix a rubber tube drain over the stump of the cystic duct and suture it in place so that it cannot get away, using the ligature on the proximal stump of the duct for that purpose. That makes the drainage-tube a part of the duct. It cannot get away. We will not put a drain in the hepaticoduodenal fossa. We fix the drain to the skin with one silkworm-gut suture. This tube will not be taken out for eight or ten days.

VISITING DOCTOR: Did you take the gall-bladder out?

DR. MURPHY: Yes; what there was left of it. I know I got it all because I began at the neck and dissected upward toward the fundus. There is nothing left of it but just a mass of connective tissue, the result of the recurrent inflammations. That tube over the cystic duct takes care of the bile that may escape, so that when the bile commences to leak, the easiest way for it to get away is out through that tube.

I will not touch the appendix because I have been working in an inflammatory field. You will note that we made a fairly large incision here. I think the remark Mr. Lane made about making the incision large enough is a very important one in the surgery of the abdomen. It does not make any difference to the patient how long an incision is made as far as the risk is concerned, and it facilitates the work a great deal.

As for the stones we found here—they did not look like stones that were left from the previous operation. If they were, they would have been in one mass and agglutinated. These are round

cholesterin stones, each about the size of a small filbert. Remember, stones have been left behind by the best surgeons, but I am sure that these stones formed since the operation six years ago. We will close the abdomen in the usual manner, suturing first the peritoneum, then the anterior aponeurosis of the rectus muscle, then the figure-of-8 sutures, and finally horsehair for the skin.

Let the record show that the gall-bladder had ruptured; that one stone was in a circumscribed encapsulated pocket outside of the gall-bladder, which communicated with the interior of the gall-bladder; that another stone was presented at the point of penetration; that a third stone had escaped from the gall-bladder and was encapsulated aseptically outside of the gall-bladder; that the lumen of the gall-bladder was not large enough to admit the end of my little finger; that the head of the pancreas had in it a mass as large as a walnut; that there was no thickening of the pancreatic duct; that no stone was palpable in the common duct; that the cystic duct was brought up and divided between two ligatures and the gall-bladder freed from below upward; that three ligatures were applied, two including the vessels alone and one including the vessels and the cystic duct; that the cystic duct was united to the drainage-tube by using the ligature as a suture to the tube, so that it remains always in that position; that the appendix was not removed because we were dealing with a septic gall-bladder; that all the work was performed in a zone of adhesions; that the omentum and duodenum were fixed on the under surface of the liver; that these were gradually separated without difficulty; that there was no hemorrhage at all.

Let the record further show that Sister reports the sponges counted and correct

If the cystic duct gets free, then the bile will leak out, but before that can happen a connective-tissue tube will be formed around the rubber tube. It will be formed within forty-eight hours, and any bile escaping from the duct will have to go out through that tube.

RADICAL OPERATION FOR CARCINOMA OF THE BREAST, WITH DESCRIPTION OF DR. MURPHY'S SPECIAL TECHNIC

HISTORY

PATIENT, female, aged fifty-seven, entered the hospital November 11, 1913, because of a tumor in the upper inner quadrant of the left breast. The tumor is about the size of an egg, regular in outline, rather firm in consistence, and somewhat tender to touch. It is adherent to the overlying skin, which is reddened, and there is a distinct elevation in about the center of the tumor. The mass is movable on the chest-wall.

The tumor was first noticed in March, 1913, by a masseuse, who, while massaging the breast, discovered a small lump in it. It was rather firm, freely movable, and not tender or painful. This mass grew gradually larger, but the patient did not pay much attention to it until two months ago (September), when she noticed that the skin over the tumor was reddened and that it was adherent to the tumor. Six weeks ago she noticed the small projection in the center of the tumor. She then consulted a doctor, who advised immediate removal of the breast.

No lymph-nodes are palpable in the left axilla. There is no tumor in the other breast. The tumor has never ulcerated through the skin. She has had no injury to the breast, so far as she knows. The nipple is not retracted, and there is no discharge from it. She was operated on for goiter six years ago. No history of tuberculosis or carcinoma in the family.

COMMENTS AND OPERATION

DR. MURPHY (November 12, 1913): What is the matter with her?

INTERN: She has a tumor of the breast, probably a carcinoma.

DR. MURPHY: Why do you say, "probably a carcinoma"?

INTERN: Because carcinoma is the most frequent tumor of the breast.

DR. MURPHY: Yes. What else?

INTERN: There is a distinct elevation over the center of the tumor. That is in favor of it being a carcinoma.

DR. MURPHY: Yes.

INTERN: The tumor is firm, movable on the chest-wall, but adherent to the skin. It is somewhat painful and tender to touch.

DR. MURPHY: *Very* tender and painful to touch. Go on, doctor.

INTERN: It has been increasing in size rather rapidly.

DR. MURPHY: What is the story of the trouble? That is what I am trying to get you to bring out.

INTERN: She never noticed the mass, but her masseuse did, and called her attention to it.

DR. MURPHY: How long ago?

INTERN: In March, 1913.

DR. MURPHY: From that time on the tumor has grown rather rapidly in size. The skin is fixed to it. It appears to fluctuate in the center. It is elevated, and it involves a large portion of the breast. Now the question comes up, did the trauma of the massage account for the rather rapid increase in size of the tumor? She has had no elevation of temperature. She has had no chills. These would not be necessary, that is, if she had a cyst primarily with infection as the sequence of the trauma, but as it enlarges it becomes more sensitive. There is no palpable enlargement of the axillary lymph-nodes. Why? Because this woman is rather stout, and it is difficult to palpate the lymph-nodes. There has been no discharge of blood from the nipple at any time. She has no retraction of the nipple.

The question that arises is, is it a cyst with infection or is it a carcinoma? It has been punctured and no fluid was obtained. The needle I use for the puncture is an ordinary medium-sized hypodermic needle. We should always keep in mind the possibility of a cyst being in the breast. These patients are sent all over this and every other land to have tumors removed from the

Carcinoma of the breast continues to reap a colossal mortality. We do not pay sufficient attention to tumors of the breast. They are usually first recognized by the family physician and he does not make it sufficiently clear to the patient that the tumor should be removed at once—*now!* We should always be exact in our differential diagnosis of these cases, and removal should be determined early. We are making strenuous efforts to diagnose and remove cancers in most difficult places in the body. We are working very hard to remove tumors by vaccines, and we are neglecting the most common and most accessible cancers—the carcinomas of the lip and of the breast. That is the proposition confronting us today. It seems to me that it will take another hundred years before we will come to a uniform practice in this class of cases.

In this case there is no retraction of the nipple, and there is a spot that apparently fluctuates; still I am of the opinion that this is a rapidly growing carcinoma and not an infected cyst.

What about the prognosis? In carcinoma of the breast the percentages of final cures have not materially changed. We would like to believe that they have, but they have not, even with our modern very radical operations. Why have they not changed very materially? Because the operation has not changed the character of the disease. Cancer of the breast is the same today as it was twenty, fifty, and more years ago.

We will encircle the breast by making two elliptic incisions extending from the outer and upper border to the lower and inner and down to the pectoral fascia. Then we lift up the breast without traumatizing the tumor.

Next I remove the aponeurosis of the pectoralis major muscle, but not the muscle because I wish to utilize the pectoral muscles for another purpose. We have this zone free now. The breast has been removed. We will examine the tumor.

It is unquestionably a carcinoma. The specimen will be sent to the pathologist at once for microscopic examination, although we are convinced that the clinical diagnosis is correct.

In performing a radical operation on the breast for carcinoma we do not take off the pectoral muscles. Unless the carcinoma

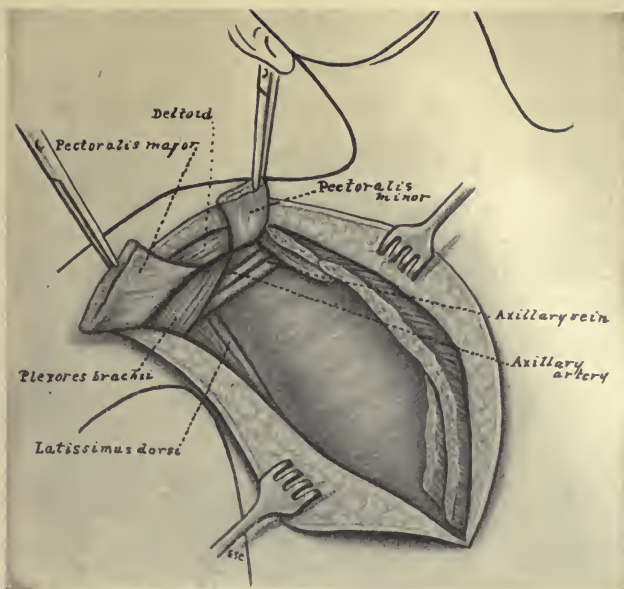


Fig. 89.—Breast operation. The stumps of the two pectoral muscles are turned upward and outward preparatory to their fixation in the axilla, which is here shown freed of its contents except the vessels and nerves.

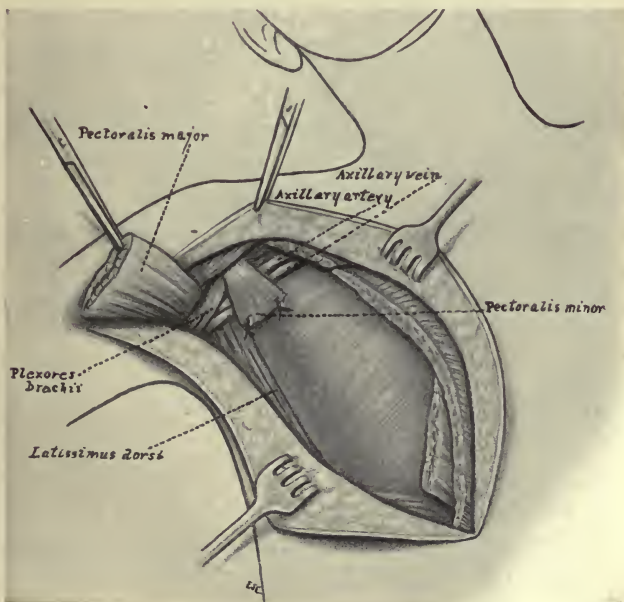


Fig. 90.—Breast operation. The stump of the pectoralis minor has been sutured to the chest-wall in front and the latissimus dorsi behind. The vessels and nerves are under the muscles.

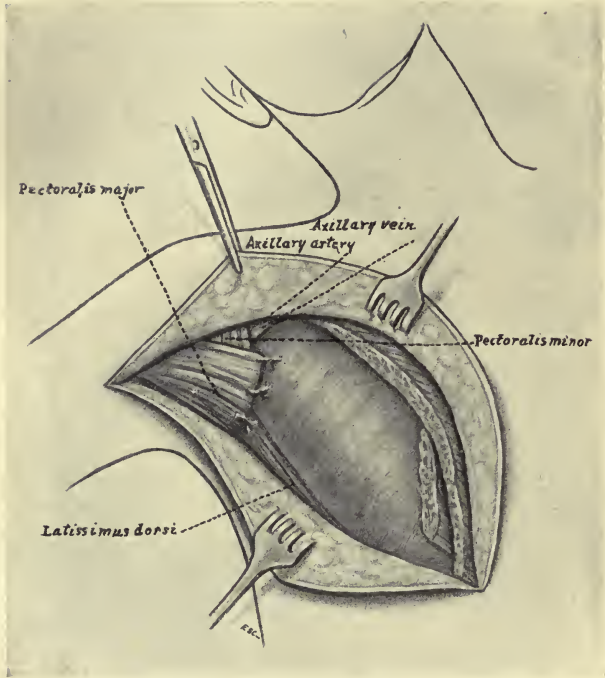


Fig. 91.—Breast operation. The stump of the pectoralis major is shown drawn down over the pectoralis minor stump, and tacked to the lateral wall of the chest with two sutures, and the latissimus dorsi with one suture, completing the filling in of the axilla and the covering of the vessels and nerves.

is on the under surface of the breast, the pectoral muscles are not involved. The lymphatics do not pass through the pectoral muscles; they are in the fascia on the surface of the muscles. Occasionally there is a lymph-node in the fat between the pectoralis minor and pectoralis major. Therefore we remove this intermuscular fascia. First we remove the anterior aponeurosis of the pectoralis major and all the fatty tissue. Then the pectoralis major is freed from its costal attachment and reflected outward on the arm, splitting it well up to the clavicle. The fascia and fat between the pectoral muscles are then removed, as a lymph-node is usually found in this location. The pectoralis minor is then detached from its costal attachment and is deflected outward. All the aponeuroses of both these muscles must be removed. Dissect free the edges of each muscle with dissecting scissors, pass two fingers under each muscle, and divide with the scissors close to the ribs, so as to leave enough muscle distally to fill in the axilla; that is, to make an axillary muscle pad. The chest portion of the muscles is elevated entirely so as to expose the fascial covering of the ribs. Remember there are no lymph-structures—vessels or nodes—in the substance of these muscles. This has been our procedure for years. When we have removed the gland-bearing tissue in the axilla and cleaned out the axilla, then we use the pectoralis major and minor to fill all that space, so that in place of having the axillary space filled with cicatricial tissue which, when it contracts subsequently, will constrict the lymph-vessels and veins, preventing a lymph return and causing an edema of the arm, there will be neither edema nor swelling of the arm, and the patient will have a perfectly useful arm. How common are the edema and swelling of the arm? You remember the splendid paper read by Greenough, of Boston, at the Cleveland meeting of the American Surgical Association, in which he gave an analysis of the cases that had been operated on in the Massachusetts General Hospital for something like twenty-five years. He found that over 30 per cent. of the cases had edema and swelling of the arm, and in some there was a total disability of the arm as a result of that compression of the vessels

by the scar tissue. Such a fearful result can be prevented by following our plan of procedure.

We have never seen a return of the carcinoma in the pectoral muscles. Mr. Bryant, of London, in commenting on that, said he had only one return in the muscles in a great many years' service in Guy's Hospital. We have not seen even a single recurrence in the muscle, so we feel safe in preserving these muscles. All the gland and gland-bearing tissue, however, should be removed.

Where do the metastases occur? First, in the axilla; second, in the second interspace, close to the sternum, just outside or inside the intercostal muscle; third, in the anterior axillary line at the sixth interspace; fourth, in the supraclavicular fossa; fifth, by the chain of lymphatics on the anterior surface of the rectus muscle to the umbilicus and into the abdomen, in the direction accentuated by Handley. He removes all the anterior abdominal wall in these cases. A fifth route of metastasis is in the skin, in a radiating manner, from the zone of primary infection.

The next proposition is, How much have these methods of operation improved the results? I am sorry to have to say, very little. If you will analyze the statistics of the greatest of the modern operations,—the Halsted,—you will find that the extent of the operation controls the final result but little; that the time of the operation is what controls the result. In place of having to do a major operation, which makes the patient appear as though you tried to amputate the upper extremity and then changed your mind at the last moment and preserved it, remove the glands at an early date, clean out the axilla, remove all the gland-bearing structures at a time when there is a chance for freedom from recurrence. Early diagnosis and early operation, rather than a late extensive operation, are the important factors in determining whether a "cure," in the strict sense of the word, can be obtained.

[Here the pathologist reported that the tumor was a medullary carcinoma.]

Now we are ready to dissect the axilla. We will take out all the lymph-nodes and lymph-carrying tissues. By going at that

from above you lessen the chances of metastases from the breast into the general lymph-system, and you are always master of the situation. We extend the incision along the anterior axillary border up onto the arm. There will be no skin scar in the axilla.

Now I am in the axilla. There is the axillary artery pulsating under my finger. We have complete exposure—just as complete as though I had removed the pectoral muscles. Now we will commence to free the fat and gland-bearing tissue from the axillary artery and vein. That is important because it is on the axillary vein that most of the lymph-nodes are situated. We “wipe” out these tissues with a gauze sponge, rarely ever using the dissecting scissors. Note that there is very little hemorrhage here. There, you see, is the brachial plexus exposed. We always carry our dissection from above downward. Now I find the glands which I could not feel before. They are on the posterior surface of the axilla, on the margin of the latissimus dorsi. We will take out the thoracic branch of this vein. We are very careful not to injure the long subscapular nerve. Why? Because that nerve supplies the latissimus dorsi, and unless it is preserved, the woman will be unable to fasten her clothes. She could not put her arm behind her body, owing to the paralysis of the latissimus dorsi. We apply the faradic current for the purpose of showing that the long subscapular nerve is all right. Now we have the axilla thoroughly cleaned out, and we proceed to ligate the bleeding points which we clamped during the dissection.

When metastasis takes place, how early does it take place? We do not know. What basis have we for saying that a case is either an “early” or a “late” one? Merely an empirical basis the result of clinical experience. Clinical experience has shown us that when the tumor is removed within a certain time, when it is small and “early,” there is no subsequent return in different portions of the body. There is nothing in an experimental way to indicate when the metastasis does take place. Tyzzer, of the Harvard Laboratory of Cancer Research, has shown that in the Japanese waltzing mice the metastasis takes place almost uniformly on the thirty-ninth day. That is the first

experimental result we have had that indicates when the metastasis takes place. We have been unable to say when these malignant cells are carried to distant positions after they penetrate the basement membrane, which gives us the right to call them malignant; therefore clinical observation is the only basis for saying that a case is an "early" or a "late" one.

Are cases "early" or "late," depending on the time the tumor is removed? Not at all. If we were to divide the cases into classes, we would say that if the patient is spare, with no loose fatty tissue, and has a tendency to the production of connective tissue for the encapsulation of the malignant cells, holding them localized for a long period of time, the chances for a recurrence are comparatively slight. The fat patient, of which this patient is a typical one, has no such tendency to the coffer-damming of the lymph-spaces and the retention of the malignant cells in the local position by the formation of cicatricial tissue. The tumor-cells pass early into the lymph-spaces, and thence into the neighboring lymphatics. They are early transmitted from one chain of lymphatics to another, and metastasis occurs early.

What is the prognosis in this case? It is very bad. First, because the woman is comparatively young; second, because she has a large quantity of fatty tissue. Women of this class do not encapsulate or coffer-dam and retain the malignant epithelial cells in their primary positions.

Where will the metastases appear? There are definite places of election. I have mentioned the routes of metastases. From the breast to the bone is the common position for metastases after the tumor cells pass out of the lymphatics. The upper end of the femur is one of the most common points of metastasis. Next in frequency is the spinal column; next, the upper end of the humerus. There is rarely ever a metastatic involvement of the smaller bones of the extremities—the radius and ulna, the tibia and fibula. Not infrequently the patient complains of rheumatism or sciatica. Within a short time you will find the size of the bone increasing rather rapidly. You will find developing in the shaft or in the upper end of the femur, in the neighborhood of the trochanter, a carcinoma. We have had a number of cases

like that. We had here in this clinic at one time five cases of metastatic carcinoma of the upper end of the femur, showing how common it is. In two of these cases the patients came in, one because she had pain in the region of the hip, which she injured in playing tennis; the other did not know she had a breast lesion at all. She had severe pain in her hip. We examined her to find the cause of the pain in the neighborhood of the hip and discovered a tumor in the breast. It did not show in the skiagram in the early stage, but while she was in the hospital it was clearly demonstrable in the skiagram that she had a metastatic carcinoma in the hip. She subsequently died from the disease. (See CLINICS, December, 1913.)

Now we are ready to place the pectoral muscle stumps into the axilla. By means of a few catgut sutures each muscle separately—the pectoralis minor first—is swung into the axilla and fixed to the chest-wall in front and the edge of the latissimus dorsi behind. Only two or three stitches are needed to do the tacking. That prevents what? First, it fills up the axillary space, and, second, it prevents the dead space from being filled up with cicatricial tissue, and subsequently causing pain, edema, and swelling in the arm. She can swing her arm in any position without interfering with the circulation in the arm. These two muscle pads cover all the vessels and nerves in the axilla. They fall into place readily. All you need to do is to fix them there. That is the only part of the technic that is original with us. We know to a certainty that we are not going to have a compression of the arteries, veins, and nerves, and that we are not going to have an edema of the arm or a neuritis. We have not hazarded the permanent cure in the least by retaining the pectoral muscle stumps. We have been doing this operation for thirteen years. We have had only good results. Sufficient time has elapsed to allow us to speak with authority on the matter.

We close the wound by putting in a considerable number of interrupted silkworm-gut tension sutures and continuous horse-hair sutures for the skin incision. There must be absolutely no tension on these flaps. We always drain these wounds to allow the wound secretion and blood to escape. A rubber tube drain

extends from high up in the axillary space downward and out through the most dependent part of the wound cavity. It is fixed to the skin at the point of exit with a silkworm-gut suture. This tube will be removed after seventy-two hours.

The arm will be dressed by the side of the body, the hand resting on the chest. A liberal dressing is applied, covering the breast and shoulder and reaching up onto the neck. A large pad is placed in the axilla. We always remove the iodine, with which the skin is painted before the operation is begun, with alcohol, to prevent an iodine dermatitis; then dust the wound with bismuth subiodide powder and seal it with collodion gauze. The dressing is put on over this.

MURPHY'S CLINICAL TALKS ON SURGICAL
AND GENERAL DIAGNOSIS

THE EXAMINATION AND ANALYSIS OF CASES

I DO not know whether any of you received the investigation report on the dispensary service of the city of New York, but it does expose the profession's dispensary work in the nicest, most quiet, genteel, yet forceful way. A committee in New York formed an organization for the purpose of determining how the out-patients in the New York hospitals were treated. I think the committee found that the dispensary attending staffs consisted of 50 or 54 per cent. of the physicians who had offices in the neighborhood. They found in the course of their investigation that a large percentage of patients in the internal medicine department never had their outer garments unbuttoned when they presented themselves for diagnosis. That tells the story. That is all one needs to tell.

The committee wrote me some months ago asking me whether I thought they ought to have paid service. I said that I thought they would obtain a higher class of and better service without pay. I must admit now that I was mistaken as far as the New York Dispensary service was concerned. They are now going to pay men to look after the free patients. That means that there will be only one, two, three, or four men now educated to practise medicine as it should be, through the dispensary school of experience, where there could be 30 or 40, if these men doing free service had the enthusiasm or the thirst for knowledge that helps to make a man when they have that large number of clinical patients at their command. I must say that I felt sad when I read it to think that they let those fine educational opportunities escape them, and every day you hear men say how much they could do if they had an opportunity—opportunities *always* exist; they are automatically made where the man is found to

take advantage of them. Opportunities commonly exceed the number of capable men.

The Johns Hopkins Hospital is contemplating adopting a pay basis for medical and surgical service. The men who will work there will be on a salary, and will not be permitted to do private practice. The same proposition is up now in Boston at Harvard. The outdoor department of the city of New York has adopted it, and the outdoor dispensary department here in Chicago will be on a pay basis inside a few years. Just as sure as it goes into effect in these places it will come to Cook County Hospital. That means that the number of practitioners who will be permitted to develop themselves in these services will be much smaller than it is now. This is the sequence of events, first, that men do not value or avail themselves of the service at its developmental value to themselves; and second, because they are not conscientious in rendering to the people the highest personal services.

What are we going to become? Just hirelings. Institutions are going to hire a doctor just as they hire accountants. Could that have occurred when Fenger, Lee, Davis, and Johnson were doing free service in hospitals? Not at all. It is time we are waking up. It is time we are cleaning house, because the people have been extremely loyal to the profession, because the profession was loyal to their best interests. It was reciprocal. I should say superlative loyalty existed on both sides. If attending dispensary doctors avail themselves of all their opportunities, they will soon become expert diagnosticians and be amply rewarded for the units of energy they expend. We find fault with the people when they go to the osteopath, the chiropractor, the faith cures, and so on, but they always come to the regular profession first, and it is only when we fail, and fail repeatedly, to diagnose their trouble and relieve it that they desert us. That is what I wish to bring out. And why do we fail? Because we do not always give them the individualization service to which they are entitled—we do not carefully examine the cases!—it is not the absence of information on the part of the profession—it is laxity in details. Think of those patients in New York who

never even had their coats unbuttoned! We try to practise on a wholesale plan, on a superficial basis.

Much of that, I believe, is being eliminated by the adoption of vaccine therapy, because that is an individual proposition all the time. We cannot resort to vaccine therapy indiscriminately or on the generalization plan. There is no average dose of vaccine nor one vaccine for all. Each case is an individual one, and we must outline a treatment for that particular case. We must use our cortical cells and not follow the label on the bottle. We cannot accept machine-made diagnoses if we are to render good service to the people.

A new type of practice is evolving. It is the men who are endeavoring to meet these requirements who are going to have the advantage. The whole profession must move in that line and move rapidly. Failure carefully to examine cases is the most common cause of error. Take a case of painful hip. Because the pain is in the hip, and because tuberculosis is the most common lesion of the hip, the doctor should not say it is tuberculosis without making a thorough analysis of the history, the etiologic evidence, and its course of development, and finally the tuberculin or laboratory test. These always tell the story if we but listen to it.

Pain and elevation of temperature, chills and sensitiveness, are not diseases. They are danger-signals and the pathologic entities must be interpreted from them. All symptoms in diseased conditions are the manifestations of a perverted physiology, or, in turn, are the result of a pathologic anatomy, and to get at the bottom of the trouble we must do more than look at the patient's tongue, take his temperature, and ask a few questions.

On what is the diagnosis to be based? The clinical course, the symptoms and signs, the physical and laboratory findings! I have often used the simile that a diagnosis is like a wheel, which is composed of a hub, spokes, felly, and tire, all in proper relationship to one another. These materials and parts separately do not constitute a wheel. They must be assembled to constitute it. The diagnosis of a diseased condition is the wheel, but is completed only by assembling all the details of the patient's ill-

ness in proper relationship to one another. First, there is the history—the story of *how* the disease began and *when* it began. *It* is the *hub* of your diagnosis. Second, the clinical course, the signs and symptoms, in chronologic order, represent the spokes and the felly. This practically makes the wheel, but you can often strengthen it or add finish by placing on it a tire, which is represented in our diagnostic wheel by the assistance we receive from the laboratory in the way of tests and microscopic examinations. (See Figs. 92 and 93.)

A good diagnosis is a scientific construction. It must be built. Knowledge, perception, painstaking care, and the interpretation of clinical history, symptoms, signs, and laboratory findings are the means needed to construct a diagnostic wheel no matter how mild or severe in degree the pathologic condition may be. The patient is entitled to the best service, and we must not only be prepared to furnish, but must render, it.

EMPHYEMA

DR. MURPHY (January 23, 1914): We have two patients, both children, in the hospital at the present time with an empyema—acute purulent pleuritis. In both cases the right side is involved, and the primary disease was a pneumonia. Empyema is a common complication of pneumonia, especially in children (over 50 per cent.), although the disease may arise by extension from the abdomen, the pericardium, or the other pleura, or it may be a metastatic manifestation of an infectious process in other parts of the body. Infection from without associated with trauma is also an element not to be overlooked. Diseases of the lung, however, most often are the cause of empyema.

In adults the great majority of pleuritic effusions are tuberculous in origin, that is, a serous exudative pleurisy. Pleurisy with the formation of fluid in the pleura is a very common sequence of pulmonary tuberculosis. Pleurisy with pus is not a very frequent complication, but is more common than generally supposed. The serofibrinous effusions are usually tuberculous in origin, whereas the purulent effusions are mixed infections, commonly pneumococcic in origin. It is

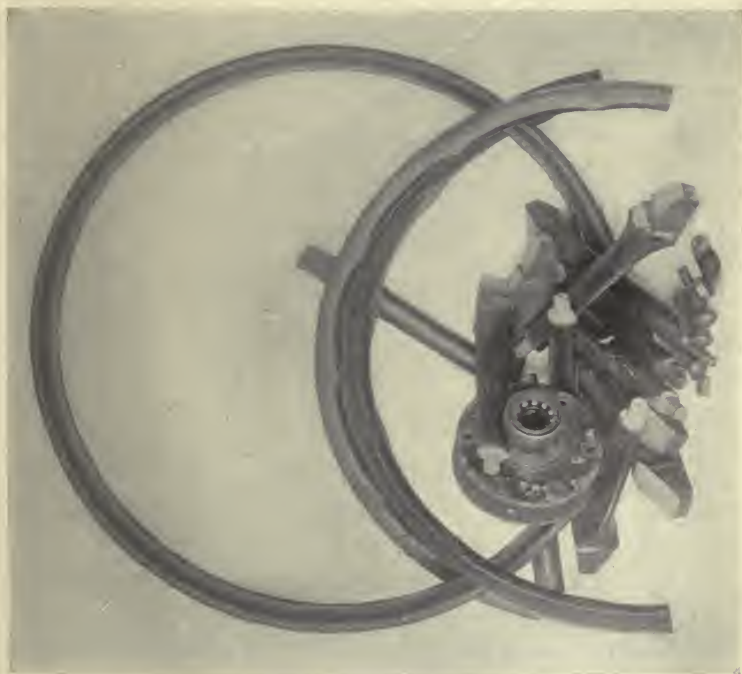


Fig. 92.—The “diagnostic wheel” as it may be visualized when the patient presents himself for diagnosis. The details of the case are not yet assembled. The history, physical and laboratory findings, are there, but jumbled together. They must be worked out in order that the diagnosis may be made.



Fig. 93.—The “diagnostic wheel” has been assembled. The hub, spokes, felly, and tire complete the “wheel,” representing each one of the essentials of a clinical diagnosis—the history, course, signs and symptoms, and the information received from chemical tests, skiagram, and microscopic or bacteriologic examination, all having a proper relation to one another.

highly probable that a circumscribed mixed infection, pneumonia, is the cause of suppuration in a tuberculous empyema. The streptococcus and staphylococcus also cause serofibrinous effusions. Mixed infections as a metastatic manifestation of pyemia are now rare, but were very common in my early Cook County Hospital experience. I have always felt that most of these were really results of miliary abscesses in the periphery of the lung. I do not believe that the metastasis takes place into the pleura or pleural cavity primarily. The microorganisms that are in the pulmonary infectious focus enter the pleural cavity through the visceral wall of the pleura. If it is a tuberculosis associated with a bronchopneumonia, you have not only the tubercle bacillus, but a mixed infection, which is producing an inflammatory area in the pleura covering that zone, whether it be pneumococcus, staphylococcus, or streptococcus. Then you have the migration of the infective microorganisms through the pleura into the pleural cavity: the pleura resists this invasion, and it rapidly fills with defensive fluid containing a large number of leukocytes, but a small number of polynuclears. It may be serous or purulent, depending on the number of leukocytes in the fluid. If it is a purulent fluid—an empyema—it may become encapsulated. Purulent fluids are more often encapsulated than are serofibrinous fluids. Only in a small percentage of purulent effusions due to tuberculosis is the tubercle bacillus found. The pneumococcus is really the most frequent organism occurring as a pure infection. It is often found in a primary empyema.

In empyema associated with tuberculosis the pus is very apt to be sterile, or it may contain other organisms than the tubercle bacillus, especially the pneumococcus.

After pneumonia and tuberculosis as etiologic factors come the exanthematous diseases. Measles is the most common, then scarlet fever, then smallpox, typhoid, and so on down the list of the so-called infectious diseases. Then we have empyema in connection with pus in other positions in the body. That is very rare at this time, but when I was a student in medicine and when I was an intern it was common. In fact, my first serious case—

my first fatal one—after I left the hospital was a man who had a Neisserian infection from which developed an ulcerative endocarditis, a purulent pleurisy, a suppurative peritonitis; practically all his joints were filled with pus, and the patient died as a result of a Neisserian infection metastasis.

The disease may come by continuity from below. The most common cause is subphrenic abscess rupturing through the diaphragm. The conditions that develop below the diaphragm that may cause an empyema are, first, appendicitis. How does appendicitis produce an empyema? An abscess may form around the appendix and the pus passes along the outer side of the ascending colon to the liver, up on the surface of the liver, between it and the diaphragm. That is then a subphrenic abscess. That abscess may rupture through the diaphragm into the pleural cavity or the pus or infective microorganisms may pass through the diaphragmatic lymph-vessels into the pleural cavity—a direct infection through the lymphatics, not a metastasis. The abscess may rupture directly into the lung. How does it go into the lung where the pleura is between? As the tissues become inflamed they tend to agglutinate, that is, adhesions form and the pleural space is obliterated. When the abscess ruptures into the lung, the patient coughs, and he expectorates an offensive pus of the same character as the pus removed from an appendiceal abscess.

An abscess around the kidney, a paranephritic abscess, may extend upward, passing between the costal arches to the cellular tissues behind. It comes up in the subperitoneal space and forms an abscess that ruptures into the lung. Remember that the kidney is behind the peritoneum. Abscess of the liver, the sequence of dysentery, was at one time a common cause of empyema, but not at all as common now, since dysentery is early controlled by diet and medication, and infected emboli do not pass into the liver.

Amebic dysentery is cured by emetin, an alkaloid of ipecac, worked out by Rogers of Calcutta. Our forefathers were not as foolish as we thought they were when they gave ipecac for dysentery. I remember a remark made by Dr. William E. Clark when

ipecac was first proposed as a treatment for diarrhea. That was the first month I started to practise medicine. Dr. Clark came into the office and said to Dr. Lee, my associate, "Are you using ipecac for diarrhea and dysentery in children?" Dr. Lee said, "No." Dr. Clark said, "Well, I have, and one of the first discharges was the doctor, but I will still continue to use it as it is a great medicine." That shows how enthusiastic that old man was in the treatment of dysentery at that time, thirty odd years ago. Now, in recent years, a man in Calcutta finds that there is in ipecac such a material as emetin, and that it has the same effect on dysentery that quinin has on the plasmodia of malaria. Think of it! After all that time! Emetin is administered hypodermatically in $\frac{1}{4}$ to $1\frac{1}{2}$ grain doses once or twice each twenty-four hours. In the liver abscess cases it should be injected into the abscess itself. It has a specific deadly effect on the ameba.

To return to the subject under discussion: An infection in the gall-bladder with rupture, or a perforation of a gastric or duodenal ulcer, may burrow up through the diaphragm. Pancreatic abscesses and pancreatic cysts may rupture and produce a rapid destruction of tissue and a purulent pleurisy. Abscess of the spleen rarely causes empyema. Echinococcus cysts in the abdominal viscera may rupture into the pleura. Infections in the anterior mediastinum or suppurative lymph-nodes at the root of the lung may cause empyema. Rupture of the esophagus is a more common cause, first, by foreign bodies; for example, the patient swallows a part of his dentistry, and it is arrested in the esophagus, ulcerates through its wall into the pleural cavity, and the pleura becomes infected. The patient has a cardiospasm or stricture, a dilatation, ulceration, and then perforation. Or, second, the doctor attempts to sound a diverticulum of the esophagus and pushes his sound through into the pleural cavity. He may try to dilate a spasmodic contraction, ruptures the esophagus, and infects the pleura. The patient has a carcinoma of the esophagus, and it ruptures through into the pleura. The patient has a diverticulum of the esophagus, and the foreign material ulcerates through the wall of the pocket and into the pleura. These are all clinical possibilities. Remember the pleura does

not at all defend itself against perforation and infection as the peritoneum does. It has not anything like the reaction to infection that the peritoneum has.

Then there are to be considered the arrest of foreign bodies in the trachea and larger bronchi, a Ludwig's angina, osteomyelitis of the ribs, although the latter condition does not frequently rupture into the pleural cavity; Pott's disease, or any infectious process in the vertebræ; submammary abscesses, where you have an abscess occurring on the under surface of the mammary gland from an infection of the gland itself, an accumulation under the breast lifting it up and which commonly perforates and destroys the intercostal muscles; then opening into the pleural cavity, and the patient has a very serious condition. Abscess of the lung wall, the rupturing of the bronchi into the pleura itself, the necrotic parasitic cysts, like the echinococcus cysts, or the actinomyces, occurring in the lung or beneath the diaphragm and penetrating through into the pleura, are all etiologic factors.

The inflammation may be general or circumscribed. The fluid not infrequently is sacculated. It may be encapsulated in the interlobar space. Other organs may become involved secondarily, such as the lung, pericardium, peritoneum, mediastinum, esophagus, endocardium, spleen, and more remote parts of the body. Cerebral abscess and thrombophlebitis are rare complications, but they have been noted. It is important to bear all these facts in mind, not only from the standpoint of diagnosis, but from the standpoint of treatment.

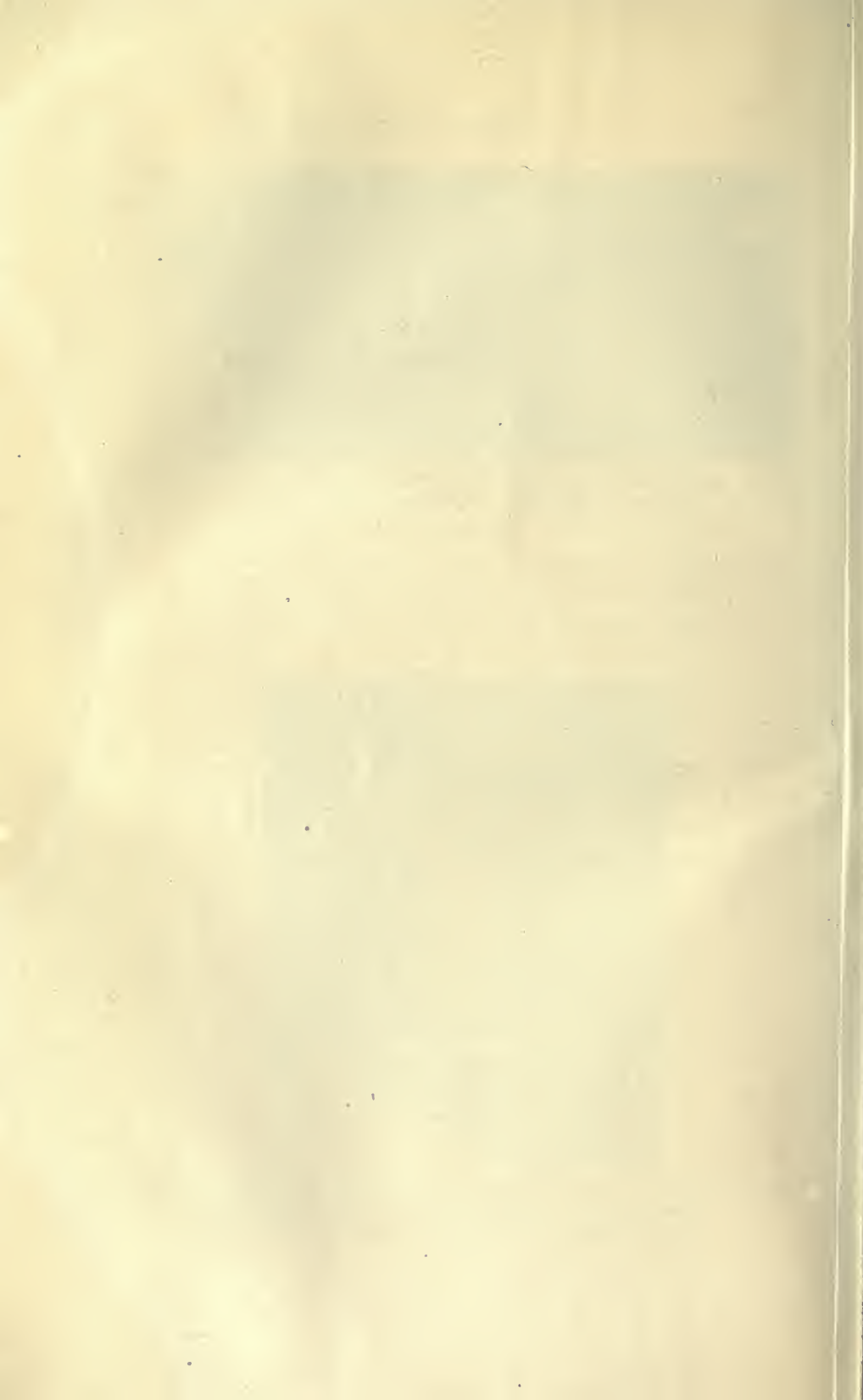
It is hardly necessary for me to consider the symptoms and physical signs, as you are doubtless familiar with them. If a pleurisy with pus is suspected, aspiration will speedily confirm or disprove the correctness of your suspicion. An exploratory puncture may be made, but remember that it is rather a dangerous procedure in cases of long standing, in which there is contraction of the affected side, with an elevation of the diaphragm and secondary suppurations in the lung, or where there is a complicating pulmonary abscess, gangrene, or a bronchiectasis. Radiography has given us much assistance in diagnosis in obscure cases. Puncture of a blood-vessel may cause a fatal hemorrhage



Fig. 94.—Osteoarthropathy in a case of chronic empyema involving the terminal phalanges of both hands.



Fig. 95.—Case of chronic empyema with osteoarthropathy of terminal phalanges of feet and hands. The typical shape of the trunk is well shown.



or a pyemia. Perforation of the diaphragm may cause a fatal peritonitis, therefore careful aspiration with a small aspirating syringe or even an ordinary hypodermic needle is safer and productive of the desired results.

With regard to the physical findings in the post-pneumonic empyemas of children, let me emphasize one point, namely, the presence of *bronchial breathing over the area of percussion, flatness, bronchial breathing through the flatness*. Put that in red ink, because that is the misleading sound. When you put your stethoscope over the flattened area, you think the patient is breathing or speaking into the stethoscope. You reason out that it is a continued consolidation of [the lung, and there is no fluid, otherwise the sound should not be transmitted; this is an error. Every man should use his stethoscope diligently when the pneumonia is not clearing up. There is the pneumonia, bronchial breathing, and bronchophony, just the same as it was on the third day of the pneumonia, but it is an entirely different condition. There is something about the acoustics of this fluid differing from a serous fluid, namely, that it transmits the sound right into the stethoscope. When that sign remains ten or twelve days after the first signs of the pneumonia, take out your smallest hypodermic needle and puncture the chest. I once asked Mr. Roosevelt how he accounted for his success. He said: "I never thought anything that other people did not think, but when I thought it I did it." That is the way with these cases. When you think there is fluid there, puncture. There is, you see, in this case, flatness on percussion, the bronchial breathing, bulging and absence of motion in the intercostal spaces, edema of the chest-wall—everything that tells that there is an effusion in the pleural cavity. Just put in that needle.

As to the treatment: Generally speaking, constant free drainage is indicated in every type of purulent empyema except the tuberculous. Sterile exudates are usually tuberculous, and if that is true, they do not need to be removed on the basis of their continued constitutional intoxication or causing metastatic infective processes elsewhere; aside from that, why should you not remove the fluid the result of a pulmonary tuberculosis?

Because the clinical history and the course of tuberculosis with hydrops and with empyema is that, while the lung is compressed by the fluid, the tuberculous process heals. That is what led us originally to the use of the nitrogen treatment of pulmonary tuberculosis, by establishing an artificial compression of the lung. While the empyema is present, the lung is compressed and the tuberculosis is healing.

In a discussion that occurred in the Academy of Medicine in Paris when Dieulafoy was speaking of chest aspiration, one of the medical clinicians said, "Doctor, that instrument has cost more lives than all the surgical instruments used up to date." He referred to the aspirator. What did this comment imply? He said, "Cases of tuberculosis will get well if you allow that fluid to remain in the chest, unless it is a purulent fluid, causing a systemic intoxication, as indicated by elevation of temperature. If you allow that to remain in the chest for a time, the lungs are healed, the tuberculosis is healed, then absorption of the fluid will commence, and the patient will get well of both his tuberculosis and hydrothorax."

That is exactly right in a large percentage of the cases, and corresponds with our experience of a third of a century. It is the immobilizing effect which the compression of the lung produces that favors the good result. Furthermore, a cold tuberculous abscess in any other position in the body should never be opened and drained, because then an additional mixed infection occurs, with little tendency to heal. It is no trouble to open the chest. That is easily done, but ask yourself always before opening it, *how is it going to close?* That is the proposition. A 2 per cent. formalin in glycerin solution may be injected into the chest to render that fluid sterile and to permit it to remain as a sterile fluid with the lung compressed until nature starts the absorption that comes with the polynuclear leukocytosis induced by the injection. That has a decidedly beneficial effect. But let me sound a note of caution: If the patient is expectorating the empyema, you cannot inject the formalin and glycerin solution into the pleural cavity because the expectoration of pus in these cases means that the process has ruptured into a bronchus. You

can see what would happen if you injected formalin and glycerin into the pleural cavity: the patient would have severe spasms of coughing. You cannot permit formalin and glycerin to come in contact with the bronchi, because it causes an irritation and an edema. The treatment in those cases is to aspirate the pus. What do you do first? Puncture with your hypodermic and then drain. Where do you drain? At the most dependent portion—at the lowest point where you can find pus.

Healing will take place by granulation, and a cicatricial contraction at the base of the cavity, beginning in the lowermost angle and extending upward, pulling the walls of the cavity together until it finally heals. That is the management when the pleural cavity communicates with the bronchi. Open and drain at once. Never attempt to treat those cases with formalin and glycerin or with any other method except open drainage.

The treatment resorted to usually in these cases of empyema is resection of a portion of one or more ribs, so that good drainage is secured. If a sufficiently large opening is made, the pleural cavity can be explored, septa broken down, and efficient drainage of pockets assured. Never scrape the walls of these cavities and never irrigate.

Thoracoplasty, the Estlander-Schede operation, and De-lorme's operation,—decortication of the lung or pleurectomy,—may be resorted to in chronic or resisting cases of drained empyema, where it is decided to remove part of the chest-wall to enable the parietes to fall in and obliterate the cavity, or where the lung is firmly bound down in the vertebral groove and the thickened pleura is removed from the surface of the lung. These operations are seldom resorted to; in fact, there would be no occasion for their performance if the diagnosis of empyema were made early and proper and timely treatment instituted. Again, it is a penalty for procrastination.

Operation: Nitrous-oxid gas is administered, and an incision is made in the posterior axillary line in the ninth interspace. As the diaphragm is pressed very low in this case, the empyema has been present for about sixteen months. After the incision of the skin the Halstead forceps is pushed through the intercostal space and

through the parietal pleura along the exploring needle. It is now spread, and you see the large quantity of gas and pus escape, as the empyema has communicated with the bronchus. Note the changes in the finger and toe tips in the skiagram and photographs, the sequence of the prolonged intrathoracic hypertension. The patient will require a subsequent Delorme or Estlander, notwithstanding his youth, and he will be permanently impaired in his respiratory exchange. A rubber drain is inserted and secured with a stitch or safety-pin. Great care should be exercised in fixing these drains, as they not infrequently escape into the pleural cavity, causing much annoyance to the patient and trouble for the doctor.

ABDERHALDEN TEST IN TUBAL PREGNANCY

History.—Patient, Mrs. J. L., female, aged forty-two, married about twenty-two years, came to the office February 17, 1914, complaining of abdominal discomfort or recurring stabbing pains in the lower abdomen. Family history negative. Previous history: Had a "*nervous breakdown*" about twelve or thirteen years ago; good recovery after three or four months' treatment. Never had typhoid or gall-bladder infection, but had severe "yellow jaundice," at the age of twenty-two, not associated with or preceded by colic.

Menstrual history: Regular and normal in every way. No pain; no flow between periods; no intermenstrual leukorrheal discharge, except occasionally after cold or exposure. Recently she has noticed that the flow was a little less in amount and of shorter duration than normal; this was for five or six months. Has never been pregnant.

Present trouble began about the middle of July, 1913, and patient attributed it to a series of carbuncles, five or six in all, coming in crops and extending over a period of four or five weeks. Since then she has not been as strong nor as well as before. About five weeks ago, at the time for her regular menstrual period, she began to flow, and for three days flowed the normal amount; she had cramps in the pelvis, which she never had had before, the cramps not being localized, but diffuse over lower abdomen.

The mild flow and the cramps continued for three weeks without ceasing. She then consulted a doctor, who gave her medicine and said that she had an erosion of the cervix. The blood flow stopped, but the leukorrhea and cramps continued for several days, when the pelvic cramps subsided, and the patient began having pain in the abdomen, which the doctor said suggested that calculi were passing from the kidney. The urine was not examined. She had difficulty in passing the urine, and passed only small amounts. It was dark colored. She vomited once at that time; there was no blood in the vomitus. She had no chills, no fever, and was not incapacitated. She drank much water and took potassium citrate. The urine became normal in amount and lighter in color.

Then she began to have daily, almost hourly, cramps across the abdomen, like gas-pains, which persisted through the day, were not referable to the taking of food, and were always worse at night. She did not vomit, was not even nauseated, but the gas pressure in the abdomen seemed very great. Her bowels were severely constipated—often did not move for forty-eight hours. Then an enema of several quarts gave immediate relief by causing enormous amounts of gas to pass. There was no blood in the stools. The “gas pain” attacks have persisted up to the present time. She had a severe attack last night, for which her doctor gave her two codein tablets. She has tried a liquid diet and a solid diet, but there seems to be no relief from the cramps. She has had chilly sensations, but never a real chill. The abdomen feels slightly distended. There is no localized sensitiveness.

Blood-pressure (in left arm) is 115 to 116. Hemoglobin is 85 to 90 per cent.

Vaginal examination by Dr. Murphy shows edema and enlargement of the cervix; the right tube is much enlarged and sensitive; considering that she had been married for many years and was never pregnant, it was proper to suspect a hydro- or pyosalpinx. The left fornix was free. The patient flowed off and on for three weeks, but not at all for the last two weeks. The mammary gland had small amount of fluid. Vulva not changed.

As the cervix was large and edematous, as it should be with a pregnancy, and the uterus free and movable and not naturally enlarged, Dr. Murphy strongly suspected a tubal pregnancy. An Abderhalden test for pregnancy was therefore ordered made by the Columbus Laboratory, which reported it "*positive.*"

DR. MURPHY: A "nervous breakdown"—when you see that at the beginning of the history it is always deterrent to a correct diagnosis, and always gives an erroneous impression. She is married twenty odd years, and she noticed that the menstrual flow was a little less in amount only recently, indicating an impending menopause. The doctor thought calculi were passing from the kidney, *and still the urine was not examined!* Five weeks from the time she had her last normal menstruation she began to have cramp-like pain in the abdomen and flowed normally for three days. Remember, she was forty-two years of age and never pregnant, although she had been married twenty odd years. But that history does not describe what that flow was—it was nothing like a normal menstruation. It was just a show for three weeks. The fact that the doctor said it looked as if calculi were passing from the kidney showed how severe the pains were, but renal calculus pains are of very short duration, and the patient is entirely free from pain between the spasms occasioned by the moving of the calculus. It is not a diminution of, but a cessation of, pain, which is so characteristic of renal calculus. When the stone is not moving, the patient has no pain. This woman had pain all the time. She vomited only once in that time. Why should the doctor call these "gas pains"? Why should a woman, forty-two years of age, without an operation or anything else, have gas pains? She had either an obstruction of the intestinal tract from a diminution of the lumen or from some foreign body in the lumen of the bowel which was continually moving or these pains were not gas pains. If these pains were not gas pains, were they ureteral pains caused by a moving ureteral calculus or something in connection with the genito-urinary tract? These are the suggestions that flash through one's mind.

What is the first thing that stands out when you are analyzing

this situation? The *irregularity* as to the *time* of menstruation and the *irregularity* as to the *quantity* of menstrual flow and the *pain* she had with it, namely, that she was one week overdue and then, when she did flow, it continued for the first three days comparatively normal in amount, and then was just a little seepage, but with recurrent attacks of hard, colicky pains.

The next proposition is, there was no albumin in the urine, no pus, no blood on our analysis, eliminating at once the proposition of a lesion involving the urinary tract. She never passed quantities of blood or mucus without a bowel movement, eliminating obstruction to the lower intestines or a carcinoma. That brings you back to what? To the consideration of what was in that uterus and what was in that tube. Forty-two years of age and never pregnant! Vaginal examination was made bimanually. (Here let me say always use two fingers, never one. You get only two-fifths as much information with one finger as you do with two fingers in the pelvis.) The examination showed a mass in the right fornix, and I assure you when I read that history that I did not put much pressure on that mass, because I suspected at once from this deviation in menstruation that she had a tubal pregnancy. She did not give a history of having endeavored to avoid a pregnancy, which is so common in tubal pregnancy, and which gives one a certain corroborative evidence that the fetus, when it has been taken from the uterus so often by abortion, finally goes higher up so that it cannot be reached. We have observed that in this amphitheater again and again. She had little or no fluid in the uterus. She had no change in the uterus—it was perfectly movable. I found no erosion of the cervix, as indicated in the history of the doctor who examined her some weeks before. There was a little redness, just as one would expect to find at the cervix, and much edema.

I believed, notwithstanding her age, that it was a case of tubal pregnancy, and I sent her direct from my office to the Columbus Laboratory to have the Abderhalden test made.

The principles underlying this test I have had placed on the board for your guidance.

Unless you appreciate the basic principles, you will never have the right confidence in it.

Abderhalden's test, the serodiagnosis of pregnancy, based on cell metabolism and protective ferments

The essentials to an understanding of the serodiagnosis are the following:

All intracellular digestion is due to specific cell enzymes.

The protoplasm of one group of cells differs from that of another chemically and physically. We once thought the nucleus and nucleolus were all important; now we recognize the significance of the protoplasm. Difference of function implies difference of structure or conformation.

Protein digestion: All proteins are amino-acid combinations. They are altered by specific cell enzymes in the digestive tract, as pepsin, trypsin, erepsin, etc., each in its successive order additionally fragmenting the amino-acid combination until they are ready for absorption and transmission to the liver.

There are two sources of nutritional supply to the blood: (1) The gastro-intestinal tract from the food; (2) from the body-cells themselves through the lymphatics.

(1) Intestinal digestive products admitted through the portal circulation are controlled, remodeled, and prepared by the liver-cells before admission to the general circulation.

(2) Cell protoplasm is foreign to the blood. It cannot be admitted to the blood without reduction again to amino-acid combinations.

The control of admission of cell protoplasm material into the blood is carried on in the lymphatic vessels, the protoplasms remodeled or controlled by leukocytes. In auto-cell-protoplasm-digestion lymphatics and leukocytes take the place of the liver in controlling, remodeling, and preparing it for admission to the blood.

Conclusion.—When the lymphatics and leukocytes of a zone are overpowered by rapid cell protoplasm digestion, substances and cells are admitted into the blood unprepared, as the enzymes of cancer, sarcoma, and placental or syncytial cells.

Abderhalden showed that the enzymes of cells are specific in the sense that they are active only against substances to which they owe their existence. He names them protective ferments. They are demonstrable by dialyzation.

Henry Schwarz, of St. Louis, has a good article in the *Journal of the American Medical Association*, August 16, 1913, where the details of the principles of Abderhalden's work are more fully considered.

When you come to figure out this case, it was about five weeks from the time of the last normal menstruation that she began to have the pain. That is very early. That would be very early to make a diagnosis of pregnancy on that test if the pregnancy were in the uterus, but whether it is early for tubal pregnancy diagnosis we do not yet know, since there have been few cases reported. But it is evidently not early, for the test was positive. I received the report at nine o'clock at night, and I called up the patient's father and told him that she should be taken to the hospital at once. As I was going away on my vacation I did not wish to take the case, so I referred it to Dr. J. Clarence Webster.

Dr. Webster examined the patient the next day,—Sunday morning,—and confirmed the physical findings and diagnosis. She was removed to the Presbyterian Hospital. She had a little more pain after the examination we made, and then the pain subsided and was less annoying until Monday. Dr. Webster made a hemoglobin test on Monday, and found that the hemoglobin was down to 54 per cent., meaning that she had had some blood leakage, as it was 85 to 90 per cent. when I examined her. He opened the abdomen, found free blood in considerable quantity, elevated the right tube, and it contained a fetus. There was a considerable quantity of red blood in the abdomen, and many clots, showing the leakage had been going on for some hours at least.

I cite this case to accentuate the importance of system when the history and physical findings apparently do not fit. Analyze the findings out to their final termination. It was the analysis of that *last menstruation* that kept me from making an error in the diagnosis that might have cost that woman her life, because

it immediately put me on guard to get her to a place where she could be taken care of immediately if the tube should rupture.

The Abderhalden principle, which is the serodiagnosis of pregnancy, will, I believe, eventually be effective in the diagnosis of sarcoma and cancer; if not in the diagnosis of cancer, it should give the reaction to the cells of the organ involved in a neoplasm, as in the liver, spleen, kidney, ovary, when in process of any active cell proliferation, as the lymphatic control over the protoplasm admitted into the blood is overpowered. The test is based on what? First let us take up the principles. The blood is fairly uniform and continuously the same. Enormous quantities of nutrient material are present in the blood under ordinary conditions. How is the blood supplied with the products of protein digestion? By protein food digestion through the portal circulation. What happens when you stop supplying sufficient food to the patient through the alimentary canal? It has then another means of supply to the cells. Where does that other supply come from? From certain cells in the system. The fat and other materials taken out from these cells are sent into the circulation, to be utilized by the cells that are active and taken from the cells that are quiescent, as during illness. These two sources of supply have definite controls, and it is on these that the serodiagnosis is based.

The cells have specific types of protoplasm. For years we considered the nucleus of every cell important; still later the nucleolus, and finally the intranuclear substance; now we have come to learn that the protoplasm is an important definite structure, and has to be acted on by a definite enzyme in order to functionate and produce a definite product. Now, then, let us take our proteins that are admitted into the stomach. They are acted on first by the enzyme-making cells of the stomach, breaking up some of the material into its amino-acid combinations. The pancreatic enzymes act on some of the molecules and break them up, and then the material passes on into the intestinal tract, where other enzymes are active, and the final product of digestion is taken up by the portal circulation and is passed on into the liver. The liver is the great remodeler of the products of

digestion that are taken up from the alimentary tract, except the fats that pass directly into the thoracic duct. The liver inhibits, reforms, remodels, so that improper material cannot go beyond the liver into the blood except under altered or diseased conditions, as indicated in diabetes. All the protein and all the starches and fats except the pure fats that go up through the chyle duct into the blood are remodeled in the liver into their ultimate amino-acids. That takes care of that alimentary tract phase of digestion.

But the phase of digestion which interests us in serodiagnosis is not that phase, but the cell digestion. Remember, I said there were two sources of nutritional supply to the blood—one through the alimentary tract and the other through the cells in the system itself. When the cells in a definite portion of the system commence to break down, their protoplasm is never admitted directly into the blood under normal circumstances, as we have thought for so many years, but the protoplasm is worked over by the lymphatics to a "blood-own" substance, or the cells call forth a ferment or enzyme which is active against the same substance from which they originated. The blood takes out of it such material as it needs to reconstruct the particular protoplasm. When you change a specific definite cell protoplasm, you change its conformation, you change its blood-supply, and you change its products. We have mentioned that frequently in connection with the flattening of the epithelial cells in the formation of retention cysts in every large gland. It does not, therefore, go directly into the blood. The cell protoplasm protein goes into the lymphatics, where the leukocytes and the lymphatic glands handle, remodel, control, and remanufacture it, so it is prepared for the blood just the same as the liver does for the proteins that come through it from alimentary digestion. We are coming to the keystone of the situation. Through the controlling force of the leukocytes and the lymphatic glands they pass into the blood system and have a definite enzyme reaction to the cells of that type only if in excess of lymphatic control.

Now, where have we a demonstration of these excessive cell changes in the body? First of all, physiologically in the syncy-

tium of the placenta. Every time the placenta starts to grow, an excessive multiplication of syncytial cells takes place and the excessive product is thrown into the blood; this blood-serum has an enzyme dialytic to human placental protein and produces peptone. It has a dialytic action, and you get what? A positive manifestation of the presence of protoplasm of this syncytium cell, which means pregnancy, and showed it in this tubal case nine weeks after the last normal menstruation and five weeks after the first appearance of the altered menstruation.

The Abderhalden test is a beautiful illustration of the results that occasionally come from patient, persistent, and conscientious laboratory work. It is a combination of many individual facts previously brought out concerning cells, enzymes, and proteins. These facts were massed together by Abderhalden to a great, scientific, every-day, practical advantage, and is a fitting reward for the ten years' labor which Abderhalden so patiently expended. It should stimulate us to encourage clinical as well as physiologic and pathologic research.

Why did I want the pregnancy test made in this case? I wanted the test not so much to make a *diagnosis* of pregnancy, which we learned to make from the clinical history, which clinical history, I think, should take precedence over all other factors, *but to confirm a diagnosis*. That is what a clinical laboratory is for. You must have a mental picture of the disease itself, that is, in its clinical manifestations; then you appeal to the laboratory for a confirmation. The result was, when we sent the specimen to the Columbus Laboratory we had the report in forty-eight hours, confirming the clinical diagnosis, that a woman, forty-two years of age, married for twenty years and never pregnant, had in her tube a fetus, because the syncytium reaction was positive and at this very early period after the implantation of the ovum. How much earlier it could have been made we cannot yet say.

Abderhalden worked on this test ten years, and had 300 cases before he made the announcement that it was a comparatively positive test for the presence of pregnancy. Now you can see how very applicable that test is going to be, not only in

the tubal cases, but in the fibroid cases in which pregnancy exists. It is so difficult to keep up with all the modern advancements that unless one has the basal facts clearly in one's mind, he is soon lost and just gets into a muddle.

I will ask the doctor to read the history over again, so as to get the fact that it was not a clear-cut picture to the man who wrote the history that pregnancy was one of the things that might be present, because the woman was forty-two and had never been pregnant; still the intern succeeded in getting hold of many of the essentials except the relationship of the last menstruation, which is so important in a tubal pregnancy. They go four and one-half, more commonly five and one-half, weeks after the last menstruation; then commence the pain and an altered menstrual flow. Then commences the recurrence of the pains, perhaps one would call it a colic, but it is the spasm of the tube as it is distending under the influence of the growing fetus within. Whether the syncytium material is admitted into the blood more rapidly when the ovum is in the tube than it is in a uterine pregnancy we do not know. That should be the case because the tube has a richer lymph-supply than the fundus of the uterus. Where there is a rich lymph supply you should get a very early enzyme reaction, as that is true of infection reactions in any organ, whether it is the uterus, the stomach, or the gall-bladder. When cancer occurs in the cervix of any organ, you have the very earliest transmission into the neighboring lymph-glands, because the cervix has a rich lymph-supply. When it occurs in the fundus of the uterus, it has a late lymphatic transmission, as illustrated in the last case I operated on in my connection with the College of Physicians and Surgeons something like eleven years ago. The patient had a carcinoma of the fundus of the uterus, and when I put on the volsella, the fundus just pulled off because it was so infiltrated; the uterus was then grubbed out. That patient is living yet. I have not had a single case of operated carcinoma of the cervix living two years without the evidence of a recurrence. The speed or tardiness of metastasis is based on the richness of the lymphatics in the affected organ. I want you to contrast the history of a similar case, which ap-

peared in the February number of the CLINICS, with this history. In the former case we had to send the patient directly to the hospital from the office. We had no syncytium test made. We had the operating-room prepared to operate on her that night in case the tube ruptured; if not, we would operate the next morning. The tube ruptured at four o'clock in the morning and we operated soon afterward. We figured out what could happen before morning and we were ready and saved the patient's life, and notwithstanding the promptness the abdomen was filled with blood when we operated and she was practically pulseless. We gave the intravenous salines as soon as I started to operate and kept up the blood-pressure. We had the tube out in a few seconds.

When you get into that bleeding tube, if you have a clean-cut idea of what you are going to do, you may save a large quantity of blood that may be vital. For instance, if it is a left tube, when the abdomen is opened, put your hand into the abdomen at the right cornu of the uterus, pass down behind the uterus and then over to the left side, roll the tubal mass forward and swing it out. It comes every time. Put your clamps on and you are master of the situation in less time than it took me to describe it. If it is on the right tube, put your hand in from the left cornu. Go down into the pelvis and get onto the tube. You know the tendency of all of these tubes when they become impregnated is to roll backward under the broad ligament. Nature tries to get it to a place where it can encapsulate it or take care of it. The moment your clamps are on the bleeding is stopped—you do not have to sponge the blood out of the abdomen; just put on the ligatures and turn in the stump and close up. The peritoneum absorbs the blood very promptly and it is an advantage to the patient.

THREE CASES OF ECTOPIA TESTIS

HISTORY

CASE I.—Master M. J. Patient, male, aged five, entered the hospital November 29, 1913, because of a mass in the left inguinal region, about the size of a hickory-nut, rather soft, and not tender to touch or painful. Present trouble was noticed at birth. On examining the scrotum it was found to contain only the right testicle; the left one was absent.

Previous history: Had whooping-cough at two weeks of age; sick for two months; had measles when two years of age, and was sick two or three weeks; no complications.

Family history: Negative to tuberculosis or carcinoma. No one else in the family has the same trouble.

COMMENTS AND OPERATION *

DR. MURPHY (December 1, 1913): We will make a straight incision, about two inches in length, parallel with and one inch above Poupart's ligament, dividing the skin and fascia down to the aponeurosis of the external oblique muscle. Retracting the wound margins, the testicle at once comes into view, resting outside of and on top of the inguinal canal. Dissecting it free by a spreading scissors dissection and then raising it up, the fibers of the gubernaculum which are attached to the anterior superior iliac spine show up splendidly. They are very well developed, much better than the fibers leading into the scrotum, perineum, or to the symphysis pubis, which accounts for the position of this testis resting anterior to the external oblique muscle and displaced an inch above the external inguinal opening. It is as good an illustration of that particular phase of faulty descent of the testicle as I have ever seen.

* As ectopia testis was discussed fully in the December, 1913, issue of the CLINICS, in all its phases, embryologic, anatomic, and clinical, only the pathologic conditions present in these three cases are considered here.

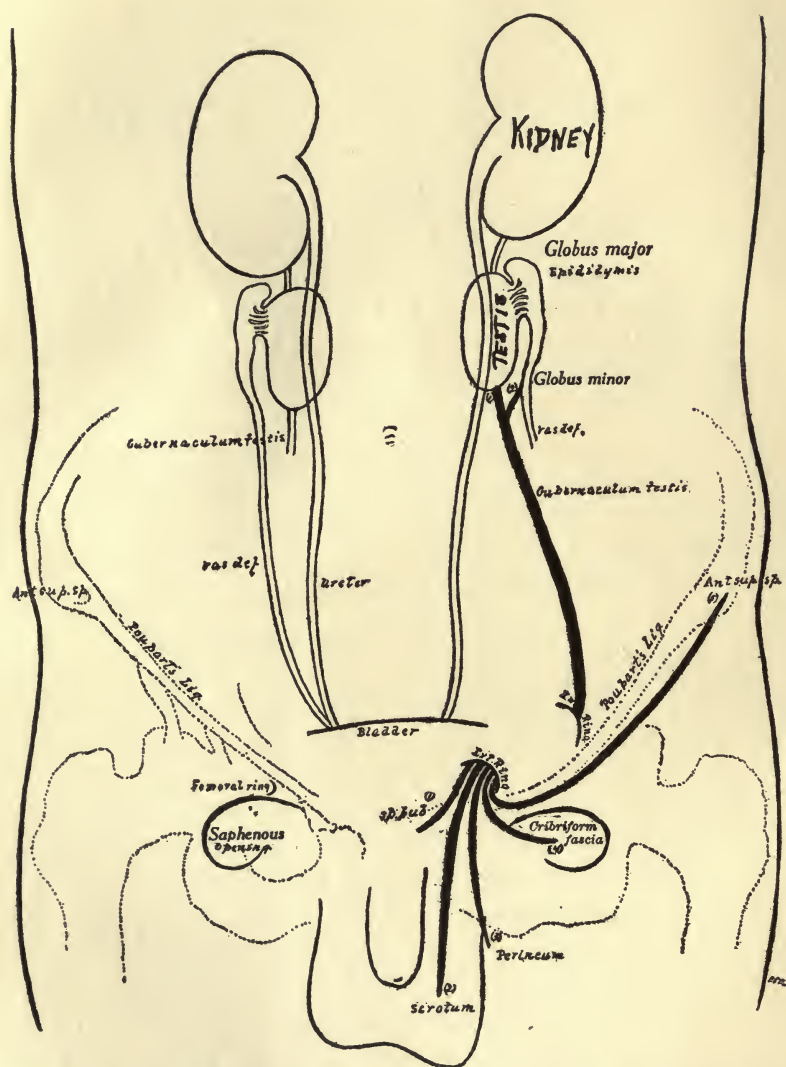


Fig. 96.—Schematic drawing of gubernaculum and course of descent of testis. The testis is attached to the lower pole of the kidney, and the gubernaculum is attached to the testis (1) and globus minor (2) above, and to the fascia of the symphysis (1), scrotum (2), perineum (3), cribriform fascia (4), and in region of anterior superior spine (5) below. The dominant line of traction determines the subsequent position of the testis.

The testicle is of about the normal size. There is the portion of the gubernaculum that goes over to the symphysis pubis, and here is the part that goes down into the scrotum. See how feeble that is! We divide the pubic and iliac spine fibers, leaving only those that go down into the scrotum, so that when we place the testis in the scrotum, it will not be pulled out of position. These scrotal fibers are usually the stronger ones, but they were not in this case. Here is the testicle proper. There is the continuation of the tunica vaginalis. We will open it on the inner side.

Let us see if that sac communicates with the peritoneum. I think it does. That looks like a portion of omentum presenting. It is. We will free it, return it to the peritoneal cavity, and close the communication.

We must be very careful in separating the vas and the vessels from this sac, to which they are closely adherent. I have now separated them. I know that the vas is free because I can feel it when I roll it between my fingers. It feels hard, like a pipe-stem. Ligate the opening in the peritoneum. Now free the vas and vessels well down along its surface and you will see they are of ample length to permit the placing of the testicle in the scrotum. I now dilate the dartos tissue with a blunt scissors and place the testicle in the pocket. A few puckering stitches are now made in the neck of the scrotal sac to secure the testicle in its new position. We now close the inguinal tract as in an ordinary operation for hernia.

HISTORY

CASE II.—Mr. L. Patient, male, aged twenty-two, entered the hospital November 22, 1913, because of a mass in the left inguinal region, about the size of a hickory-nut, rather firm, not painful or tender, freely movable, and giving some impulse on coughing.

The present trouble was first noticed at the age of fourteen, when the patient would have nocturnal emissions two or three times a week. He consulted a doctor, who found that the left testicle had not descended. When lifting or shoveling, he has pain in the left inguinal region. The left side of the scrotum does not contain a testicle.

Previous history: Had pneumonia at one year of age; no complications.

Family history negative to tuberculosis and carcinoma.

COMMENTS AND OPERATION

DR. MURPHY (November 24, 1913): This man has a mass in the left inguinal region about the size of a testicle, not painful

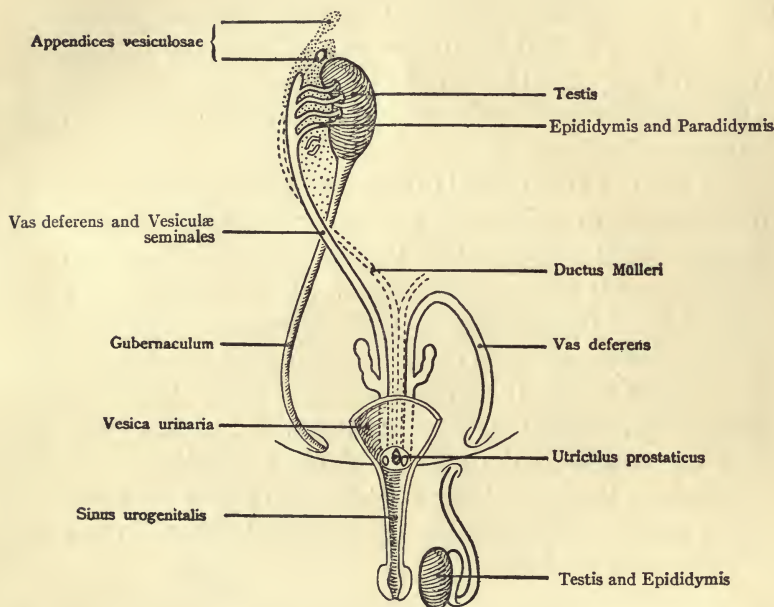


Fig. 97.—Shows the embryology of the male and female generative glands, and the result of testicle descent to the scrotum (Kollmann).

or tender to touch, and on examination of the left scrotum there is no evidence of a band there which represents the gubernaculum testis. Not only that, but the scrotum on that side has only a thin skin covering. There is no testicle in his scrotum, nor is there dartos tissue in the scrotum. The skin is there, that is all.

He has had a descensus of the testicle through his inguinal canal entirely, and he had dartos tissue formed around the testicle just outside of the ring, but in place of going into the scro-

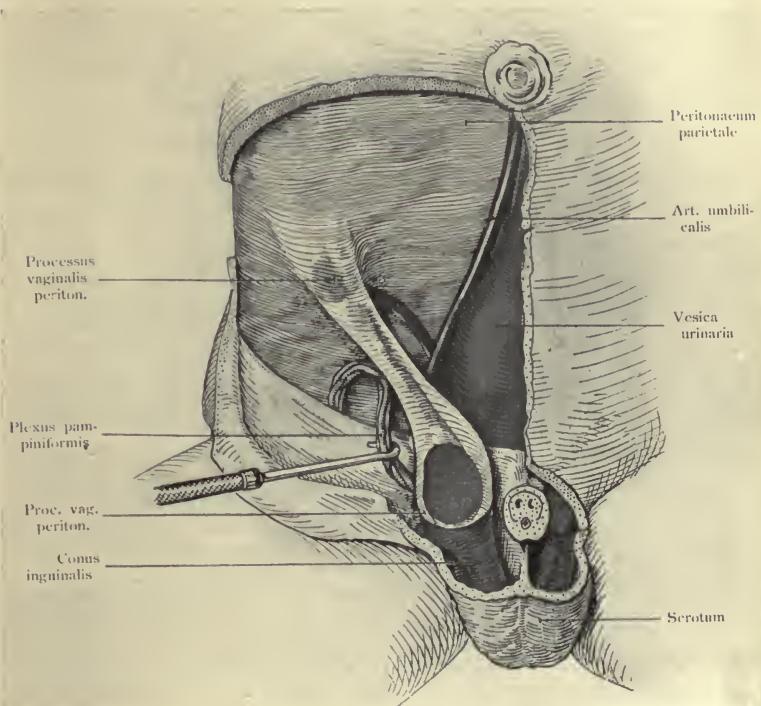
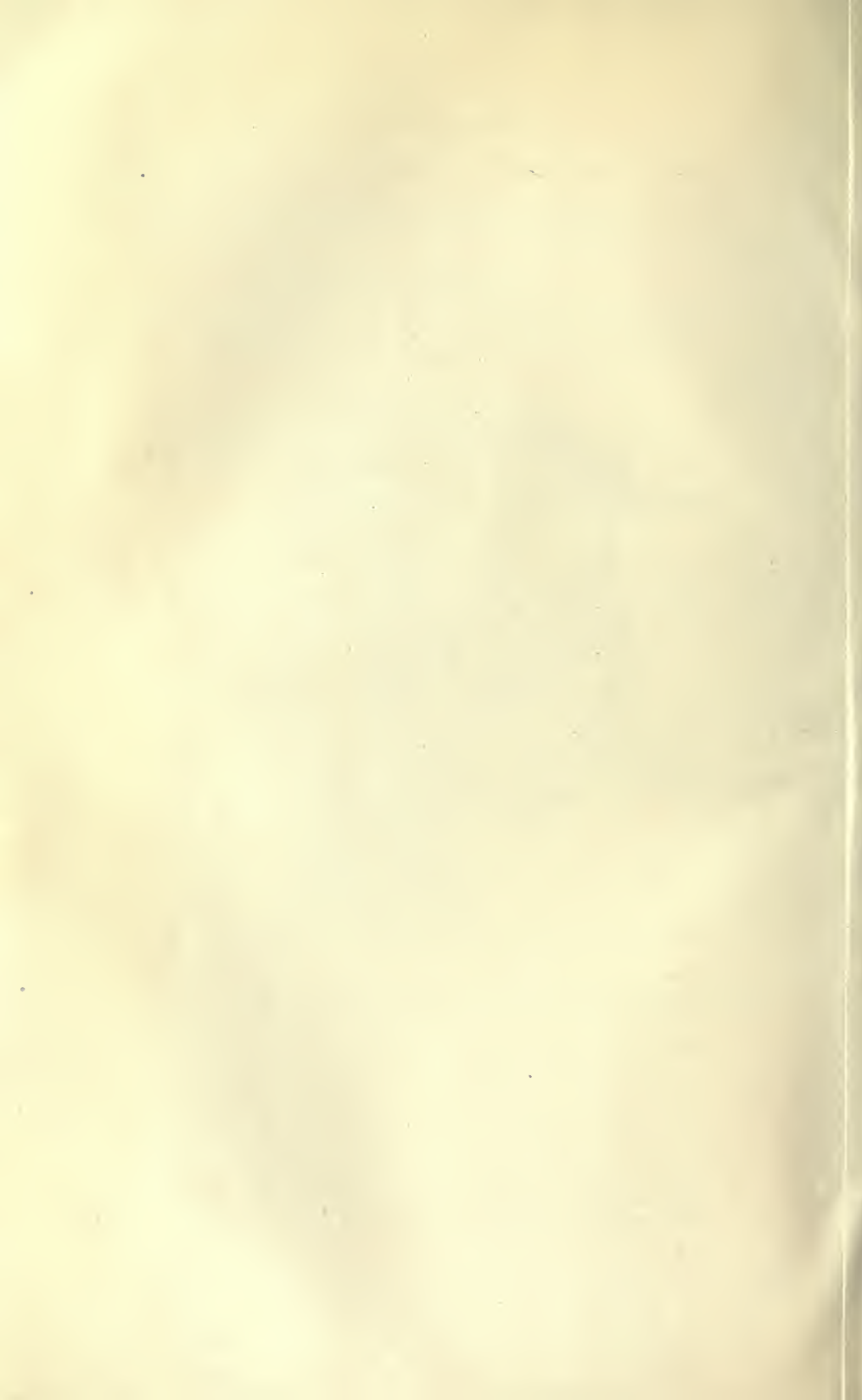


Fig. 98.—Shows the gubernaculum and the processus vaginalis in the evolution of the tunica vaginalis, also the pampiniform plexus of veins (Kollmann).



tum, it went up onto the abdominal wall and is external to the oblique muscle and to Poupart's ligament.

I had a boy who had an absence of the dartos on both sides, the dartos remaining as a thick elevation above the pubis, making a *mons veneris*, so that when you came to handle that case, you had not only to deal with the production of a pocket, but you had to take down all that dartos tissue and transplant it into the skin of the scrotum. We secured a perfect result. Five years after the operation you could not tell that he ever had any trouble with the testicle, because we had transplanted his dartos down into the scrotum with his testicle. If we had endeavored to dissect the testicle free from the dartos tissue and bring it down, then he would not have had a scrotum of normal appearance at all. He would have merely a testicle inside of a skin capsule, with absence of connective tissue. Therefore I shall transplant all the dartos tissue in this case from the position on the anterior abdominal wall to the scrotum.

We make the usual incision and expose the mass, which, you see, is the testicle. There is no dartos there at all on this side—just skin. Now the question is, whether he still has a congenital communication between his peritoneum and the tunica vaginalis in its malposition. We do not know. We do not know how the veins of the pampiniform plexus are distributed here. We have the testis fairly well freed down to the aponeurosis of the external oblique. Let us see whether it has come out above or below, whether it came out through the inguinal canal and went up, or whether it came out directly through the wall from the internal inguinal opening. There is the testicle, the dartos tissue, and everything that goes with it. It came out through the external inguinal ring and then went upward.

The next question is, has he a hernial sac? I think he has. See that white line! There are veins passing along close to it. We must get this testicle free in front, clear back to the neck of the canal, so that we can displace it to where it belongs in the scrotum. There is a little loose tissue at the base where the dartos should be. There are the veins of the pampiniform plexus. Now the question is whether that white sac that we see there is

the tunica vaginalis and with what does that tunica vaginalis communicate. That looks as though it is a hernial sac. Here is the testicle. Let us see if it will go back into the abdomen. It will not. The question is, is this sac occluded? Is it separated from the free peritoneal cavity? If it is the sac, it should be on the inner and upper side, just the same as in an inguinal hernia. Here is the cord going back here. There is watery fluid coming out. There is the peritoneum, as it appears to go through from the peritoneal cavity. Let me free that. Let me see if I can separate that from the vas and the vessels. Let me see if there is another pocket, another line of communication. There is a communication downward you see. There is the free cavity. Let us open that and see whether that goes into the testicle or into the peritoneum. That looks like a hydrocele in the canal of Nuck in the female, but it is on the cord in this case. Yes, there is a hydrocele. There is no communication between that and the peritoneal cavity. We must separate this from the vas and the vessels. There is the portion of what was intended originally for the tunica vaginalis; I will completely free it from its position above, because otherwise we cannot bring that testicle down as low as we desire. We will later make a typical repair of the hernial canal on the Andrews imbrication plan.

The next step is the displacement of this testicle down into a very small scrotum, which is done as in the preceding case. That was a very pretty case. What are the chances of a malignant degeneration? None, I think, as this is a complete, perfectly developed testicle. The chances of malignancy are greater when they are aplasic and external. This testicle appeared to be fully developed, but the gubernaculum drew it in the wrong direction when the testicle came outside. It is not so much a lesion of the testicle as it is a lesion of the peritesticular tissue and a displacement or faulty development of some of the fibers of the gubernaculum.

Let the record show that it was an ectopic testicle which came through the normal canal, and then ascended on the abdominal wall on a line apparently with the inguinal tract; that it was subcutaneous with the dartos tissue; that it was freed at

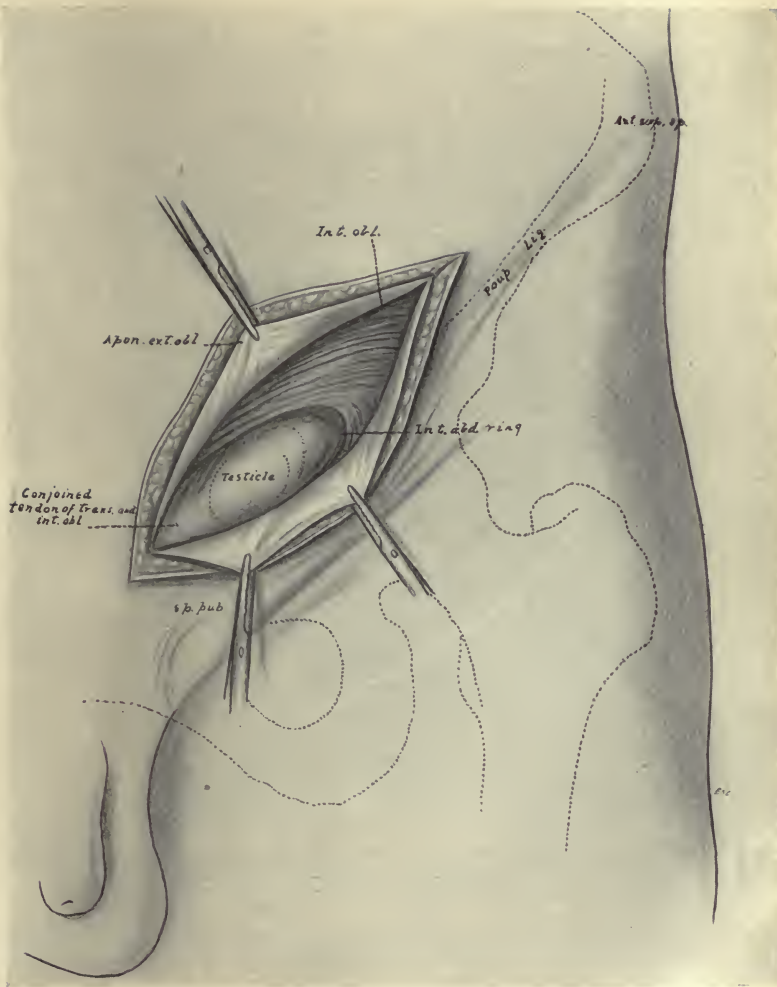


Fig. 99.—The external inguinal ring is exposed, the testis lying in the ring. The aponeurosis of the external oblique has been split, the fibers of the internal oblique presenting in the wound

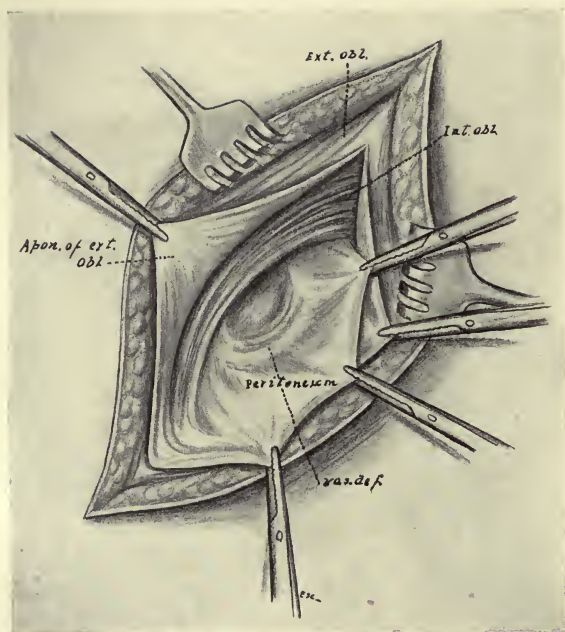


Fig. 100.—The peritoneum is drawn out preparatory to its incision. The vas is shining through it.

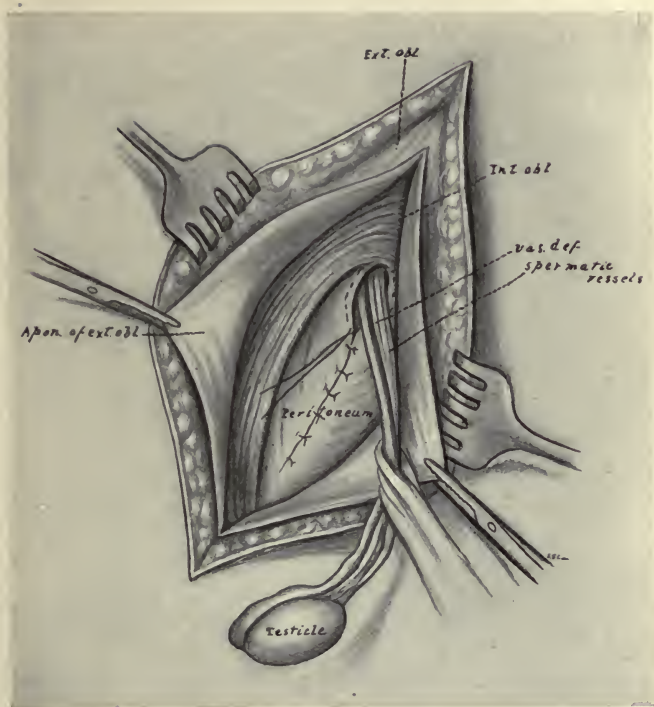


Fig. 101.—The testis has been freed, the fibers of the gubernaculum divided, and the testis is ready for its transplantation into the scrotum. Also the closure of the floor of the canal.

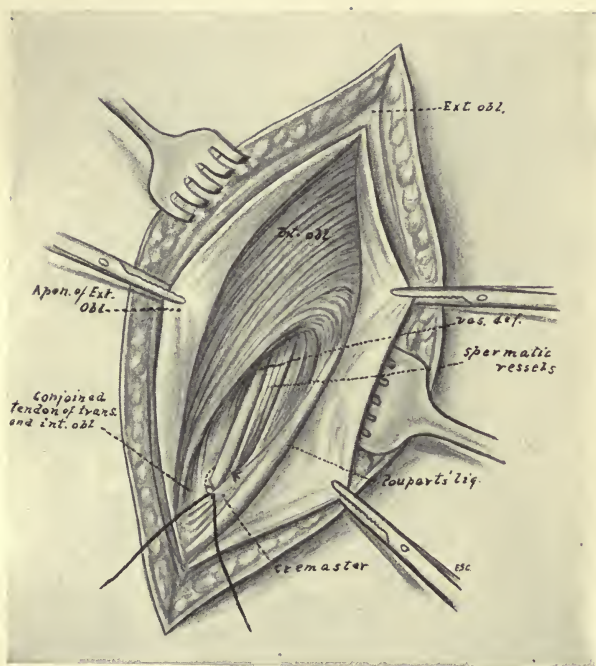


Fig. 102.—The testis is replaced in the scrotum and two catgut sutures have been placed in the lower angle to strengthen it.

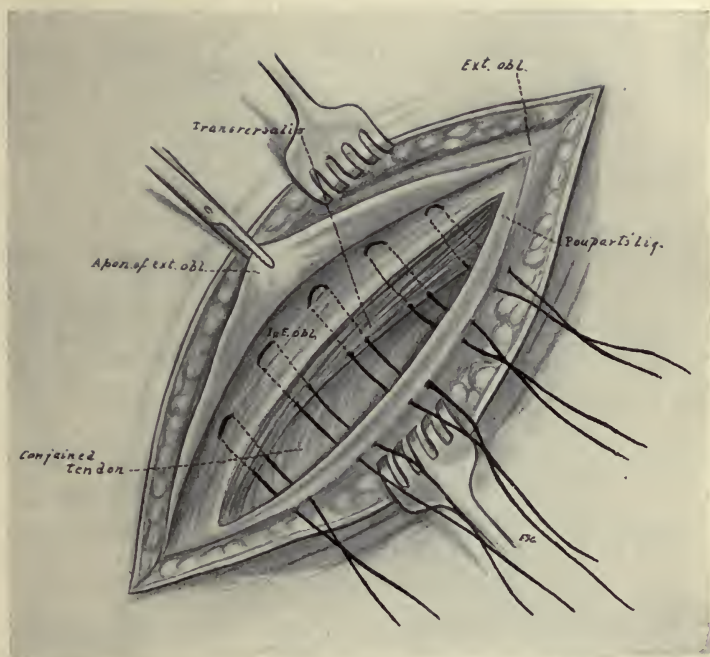


Fig. 103.—First step in the imbrication of the external oblique aponeurosis flap. The upper free edge is to be drawn underneath the lower free edge when the five sutures are tied to form the floor of the new canal.

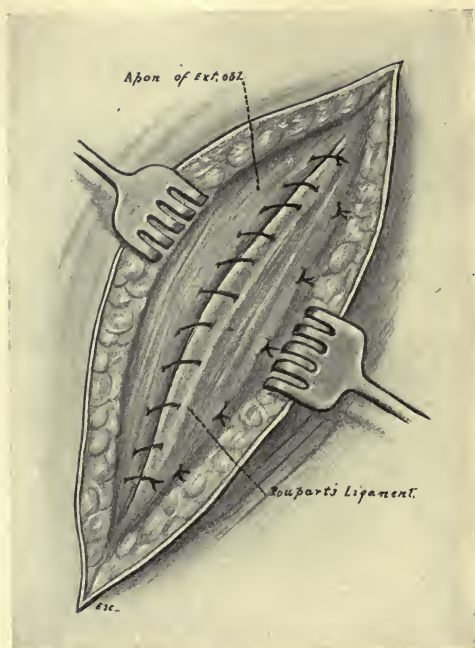


Fig. 104.—The imbrication is completed. The free edge of the lower flap has been sutured to the aponeurosis of the external oblique to form the roof of the canal. The five knots show the location of the edge of the upper flap.

its upper pole, the canal and the lower portion of the dartos exposed; that there was no communication between the tunica vaginalis and the peritoneum; that there was a separate hydrocele of the cord.

HISTORY

CASE III.—Mr. R. Patient, male, aged twenty-three, entered the hospital November 24, 1913, because of a small tumor about the size of a hickory-nut in each inguinal region. These tumors are not painful or tender to touch, are freely movable, and impart some impulse on coughing.

At the age of fifteen (eight years ago) the patient first noted that his scrotum did not contain testicles. It was merely a small empty sac. In the last few years he has been nervous, with frequent attacks of twitching of the extremity muscles. His bowels have been constipated for the last five or six years; previous to this time they were regular. He does not have any pain in the inguinal regions after exercise, walking, or straining. The scrotum is contracted and small, and does not contain either testicles or dartos.

Previous history: Had pneumonia in 1906, lasted six weeks; no complications. No venereal infection.

Family history: Negative to tuberculosis or carcinoma.

COMMENTS AND OPERATION

DR. MURPHY (First operation, November 26, 1913): This is a very interesting case. I think we have had every conceivable deformity of the testicle in the last four years, every one described in the literature, and some that are not. One was a hermaphrodite; the patient had a well-developed penis, fairly large bifurcated testicular sacs, a canal leading through a narrow vagina to a small-sized uterus, and well-developed tubes and two testicles, both attached to the broad ligament near the ends of the tubes. One had undergone sarcomatous degeneration and on microscopic section it was a round-cell sarcoma in a tissue histologically testicle.

We make the usual incision as in inguinal hernia here to expose the testis, and here it is. It started to come down from the

internal ring, and then was arrested midway between the external and internal rings. There is the gubernaculum. See how firm these iliac fibers are! There is the testicle, and there is the tunica vaginalis. We must free that so that we will be able to place the testis down into the scrotum. It is malformed. The epididymis is not attached in the regular way. It is attached to the upper pole of the testis only. We must cut these fibrous bands extending to the superior-anterior iliac spine, otherwise that testis will not come down. See the strength of the internal oblique where the cremasteric muscle should have formed and gone down, but did not!

There is the vas on the inner side. It is much smaller than normal, and, as usual, firmly adherent to the peritoneum. Now, you see, I am under the peritoneum, and I have separated it from the vas. I am going to close the peritoneal cavity. I am ready to tie that sac, but before I do so I must spread this tissue with care, because the vas is such a small affair in this case that it is entirely out of proportion. I have nothing in the ligature except the peritoneum; nothing in or outside of the peritoneum is ligated. I will divide the tunica and permit this lower portion to go down and the upper portion to go up. That relaxes my tissues very much. Now, then, we will commence to pull the cord upward and let it come gradually until I get it freed enough to permit of its easy displacement into the scrotum. Look! I am wiping the peritoneum from the vas and there is the vas shining through like a white cord. There is the testicle. See how perfect it is, except the absence of attachment of the epididymis to its side. I have not disturbed the spermatic artery or the vas. Now I will tie this opening in the peritoneum, so that we have a complete closure of the peritoneal cavity.

Now we are going to bring the testicle down into the scrotum. This man has practically no dartos tissue at all. There is skin there, but there is no dartos in the skin. I am going to bring that down before placing the testicle into the pocket I will make for it in the scrotum. I will fix it there with a few catgut sutures so that it cannot possibly recede.

I think, all things considered, that it may be good surgery to

operate on only one of these testicles today. If that survives, as it should, then we will be able to take greater chances, if necessary, with the other one to get it down. If it should not survive, then we will do less hazarding of the other testicle in the second operation.

Let the record show that the testicle came directly out from the internal ring and rested in the canal between the internal and external oblique muscles; that the gubernaculum did not come down to the external ring and did not pass into the scrotum; that there was practically no dartos tissue; that we freed the testicle and opened the peritoneum; that we separated the vas from the peritoneum well down into the pelvis, so that we had a perfectly free testicle; that when that was freed, the scrotum was prepared and a pocket made in it for the reception of the testicle; that the scrotum was dilated with an eight-inch angiotribe; the testicle then was perfectly free and displaced into the remnant of the scrotum; then it was secured and fixed by a puckering suture at the level of the lower end of Poupart's ligament; the cord was brought down under the aponeurosis of the external oblique, which was overlapped by the imbrication plan with a single suture; there was no tension on the vas or vessels when the operation was completed. No sponges were used in the peritoneal cavity, and therefore no record is made of them.

This was a case of direct transmission of the testicle through the internal ring to the middle of the inguinal tract, differing entirely from the case we had on Monday, where the testicle came through the external ring and passed along upward on the abdominal wall in place of passing down into the scrotum. Note that the epididymis did not pass back over the testicle in the usual way. The epididymis may remain attached to the upper pole of the testicle; second, it may be attached to the upper pole or to the lower pole or at the hilum; third, the epididymis may be entirely detached, not fixed in any way either to the vasa efferentia or to the testicle proper; fourth, the testicle may be without an epididymis and without the vas, attached to the tubes or to the connective tissue resembling the tubes. These latter are hermaphrodite types. One of the greatest

dangers of the cryptorchid is that the testicle may become sarcomatous, or if he lives to a more advanced age, it may become carcinomatous. It was believed for a long time that the trauma of passage in and out of some portion of the inguinal canal was conducive to that malignancy. In the case of hermaphroditism cited, the testicle was not traumatized at all. I believe the point made by the embryologist that aplasia or failure of complete development of the testicle is conducive to sarcomatous degeneration is true.

Occasionally you will find that the testicle is situated between the muscles of the abdominal wall and that the spermatic artery comes from the common iliac or from the aorta, as high up as the renal artery. Where that occurs you are unable to bring the testicle down unless you divide the spermatic artery, but it is fairly well agreed that it may be divided if the veins are protected; and if you do not traumatize the vas and the vessels around it, they will be sufficient to maintain the vitality of the testicle if it is transplanted into the scrotal region in that position without fixation and without pressure on the vas, as was ably demonstrated by Bevan.

The results in these operations are fairly good. We have had no unpleasant experience, as necrosis of the testicle, in this transplantation. We have had descending thrombosis of the spermatic veins following operation for varicosity, with a subsequent necrosis of the testicle and atrophy, without suppuration, but this is very rare.

(Second operation on opposite side December 13, 1913.)

We operated on the other side a few weeks ago, and we have a good result. See! there is a good-sized testicle in the scrotum surrounded by dartos. We were able to bring the testis down and keep it down.

I make my incision high on the abdominal wall, three-fourths inch above Poupart's, just as for a hernia. I want to expose the aponeurosis of the external oblique all the way. Then we will expose the inguinal tract; there is the aponeurosis of the external oblique overlying the gland; it is divided. Now the testicle is free. It is external to the internal oblique muscle. There is the

dartos tissue resting around it. The internal oblique muscle and the continuation of it that goes to make up the dartos and the cremaster muscle fibers with the conjoined tendon are behind the testicle.

See what a large testicle! It should be a normal functioning one. It rests in the canal outside of the peritoneum, with all its tissues apparently in position; one does not often get as nice a picture as that. There is a mass of the dartos spreading toward the symphysis, and here is the portion of the gubernaculum that goes up to the anterior superior iliac spine. Now we are down to the peritoneum, and here is the tunica vaginalis. Let us see if that communicates through the canal with the peritoneum; it is apparently closed. There is the fibrous band that prevented this testicle from coming down; it represents the continuation of that peritoneum; it is a white and clean-cut cord. I must separate it from the vas and the vessels to admit their descent.

I must free the vas and the vessels by a spreading dissection and get between the peritoneum and the vas and the vessels; it can best be done from behind.

Now we will close the peritoneum as the vas is extensively freed from it. There! the sac is free. Now we are ready to put that testicle down where it should be without traction on the vessels or the vas. The greatest danger is in tying the bladder outside the sac, as it is often fixed to it. You make the sac a part of the ligature by putting in the puckering stitch of the ligature outside of the constriction in the sac, just the same as in a hernia; you see the ligature and the sac are one and the same. This cannot get separated.

The next step is to put the testicle down into the scrotum. Let us get the scrotum exposed and see how I can handle the scrotum and just what I am going to do. I pass down into the scrotum an eight-inch clamp, and by spreading it I make a pocket for the testicle. This band of tissue has practically no vessels in it, because it is just a continuation of the gubernaculum that has gone over to the symphysis and has not gone down into the scrotum, where it should have. Now take this testicle and

put it down into the scrotum, and constrict the neck of the opening with a purse-string suture so as to get all the tension off the cord and still not constrict it.

He has no hernia, but a tendency to the formation of one exists through the defect now in the canal; therefore we will do a typical Andrews imbrication operation.

Let the record show that the testicle rested in the canal and external to the internal oblique and transversalis; that it carried down a diverticulum of peritoneum in the direction of the stump of the tunica vaginalis; that after first freeing the testicle and its dartos and the surrounding vascular tissue, we found that the testicle was very well developed; that its epididymis was attached to it in the normal manner, and that it should continue to functionate as a normal testicle. That we prepared a pocket in the scrotum by a dilatation with an eight-inch forceps, and put the testicle in and secured it first by two sutures to the connective tissue at the base of Poupart's, so that it could not strangulate the vas or the vessels and still retain the testicle in the scrotum; that we then reconstructed the inguinal canal on the plan of the overlapping flap or imbrication operation.

[Healing per primam in both sides, and four weeks after the operation one would not know he had ever been a cryptorchid so perfect was the anatomic restoration.—ED.]

CHOLELITHIASIS; PERICHOLECYSTITIS; STONES IN CYSTIC DUCT; CHOLECYS- TECTOMY

HISTORY

PATIENT, female, aged thirty-four years, entered the hospital December 1, 1913, complaining of pain in the epigastrium.

Patient states that four years ago—September 23, 1909, at 4 P. M.—she had pain in the epigastrium, followed by one attack of vomiting. The pain persisted for three or four hours. A plaster was applied to epigastrium and she went to bed. By 9 P. M. she was free from pain. She did not remain in bed the next day.

October 16th, 1909, at 2.30 P. M., she had the same pain, followed shortly by vomiting, which eased the pain. By 6 P. M. in the evening she was well. The patient states that about once a month she would have these attacks.

Friday, May 21, 1913, at 2 P. M., she was seized with a severe, intermittent pain and vomited many times until midnight Saturday. She remained in bed until Monday evening. In the former attacks it was only necessary to lie down for several hours, but this time she was in bed two days. This has been the case since May. During the minor attacks, hot water by mouth apparently gave her some relief. Two weeks ago she had an attack of severe pain and she vomited for three hours.

Present illness dates from Friday, November 28, 1913. At 8 A. M. on that day the patient suddenly became ill one-quarter of an hour after eating. She had a sharp pain in the epigastric region, radiating through to the back, followed by nausea and vomiting at 11 A. M. The pain then localized itself in the epigastrium. At 4 P. M. she vomited again, this time a greenish material. This condition continued almost all day Friday, and with remissions on Saturday. Saturday night she had a severe

pain. Sunday noon she felt better, but at 4 P. M. the pain returned, as did the vomiting at 5 P. M. One hypodermic of morphin relieved the pain. At 3 A. M. Monday she vomited again. At no time was she jaundiced. Temperature ranged from 98° to 100° F. No chills.

EXAMINATION BY DR. MURPHY: Perpendicular finger percussion over the gall-bladder showed the presence of tenderness. Hooked-finger palpation under the right costal arch was not particularly painful to the patient.

COMMENTS AND OPERATION

DR. MURPHY: The hooked-finger pressure under the costal arch we described many years ago as a test of gall-bladder disease—that is when the gall-bladder is distended and tense. Hook your fingers under the costal arch and ask the patient to take a full inspiration, then with the other hand strike the flexed fingers just at the height of inspiration. If pain is elicited, it is a positive sign that the gall-bladder is distended. That is not as good a test, however, as the perpendicular finger percussion test. That is the best test of all. Place your middle finger, held straight and rigid, at the tip of the ninth costal cartilage, and ask the patient to take a full inspiration with the eyes closed. When the height of inspiration is reached, strike a sharp blow on the finger. If the gall-bladder is distended or the seat of an inflammation, the pain elicited is severe. You can always make the differential diagnosis between a distended gall-bladder, a urinary calculus, and an appendicitis in that way.

I think that these percussion tests are the most valuable contributions I have made to medicine and surgery in the way of aids to diagnosis. They are so characteristic in the early stage that one can scarcely err. In cases of obstruction to the ureter we resort to the fist percussion over the kidney zone; if you have an obstruction to the cystic duct, resort to the perpendicular percussion or the hooked-finger test; then the deep pressure bimanually over the appendix. You can make the differential diagnosis at the bedside with these tests. You do not have to go home for your instruments; you do not have to have a blood

count made. You just have to use your brain and your fingers. The proper application of both of these gives results and positive results. [See CLINICS, June, 1912, for a complete description of these diagnostic methods illustrated.]

To go back to this clinical history: it extends over a period of four years—four years of recurring attacks of pain. We did not see her in an acute attack, so we were unable to make the finger test then. We saw her after the acute attack had subsided. She never had a jaundice. She says that the sensitiveness has always been on the right side. What about the urinary findings?

INTERN: They were negative on admission.

DR. MURPHY: The urinary findings are important, because unless you see these cases in the acute attack, you may err as to whether it is a stone in the kidney, an appendicitis, or a gall-bladder lesion. If it is a kidney case, you always have blood in the urine for several days after the acute attack has subsided. In these gall-bladder cases there is a history of these pains coming on, lasting a considerable time, then slowly passing away, but leaving always a soreness for a couple of days after disappearing. That soreness is not common with renal calculus. The rule is with renal calculus that the patient is perfectly well immediately after the acute attack has subsided. With appendicitis the rule is that several days elapse before the soreness disappears.

That recalls to mind the time when a doctor got me out of bed at 6.30 Sunday morning to see a case of appendicitis, supposed to have begun in the night. I felt that he could have called me at nine o'clock just as well, especially on Sunday, when Saturday is my hardest day. Let me give you the story because it was important, and the story was the thing that kept me from making an error. The physical findings were those of an appendicitis. The appendix was particularly tender on deep pressure. The patient resisted bimanual percussion, and he was sensitive over the kidney, which was suspicious. Then he told this story: He had been motoring down the Hudson, and somewhere between Troy and Albany one afternoon he got this attack of pain in his side. It was so severe that he had to stop his car at a farm-house and go to bed. The lady applied hot stove-lids wrapped in

woolens to him. Within two hours he was back in his motor going down the river. Appendicitis does not occur that way. Appendicitis does not subside that way. It was just that portion of the story that kept me from making an error.

He had been brought in an hour or so before I sent the urine to the laboratory to be examined. The report came back "full of red blood-corpuscles." That confirmed my suspicions, namely, that it was a case of renal calculus colic that he had on the road. We kept him in bed, put on hot applications, the pain subsided, and the third day after he passed his renal calculus. That was a case where one was likely to err if he did not have a system, going over the history and checking it up with the clinical findings.

This patient before us today had no temperature elevation in this last attack. She had no increase in the leukocytic count. There was fist percussion sensitiveness over the gall-bladder, as far as we were able to learn from the report, which shows that it was not a kidney lesion and not an appendicitis. Each of the previous spells subsided as that one subsided. It can and does occur in connection with a cholelithiasis, unless they have a sepsis, that there is practically no temperature elevation with the attack. Nor is there an increase in the leukocyte count. They always have the sensitiveness to perpendicular percussion over the gall-bladder, so you can make that differentiation the first time. A stone may be located in the body of the gall-bladder or in the neck of the gall-bladder all this time, or it may be passing on down through the cystic duct, but remember that a foreign body in a duct causes a colic only when it is in transit. Just as soon as it becomes stationary the colic ceases, and the patient is free from colic until the stone starts to move again. The stone may remain in position for a month or a year and not cause the slightest colic, but when it begins to move the patient has colic. That is just the same whether it is a stone in the intestine or in the ureter or in the cystic or common duct. As soon as it ceases advancing, it ceases to cause colic.

You will remember the story, related in one of the CLINICS (April, 1913), of the man who had eaten plum pudding when he was a boy, and we took it out of the intestine twenty years after.

He had innumerable attacks of intestinal colic and obstruction. The plum pudding would move along and cause colic and obstruction until it got into a diverticulum. Then the colic would cease, the feces would go by, and he was over that attack of obstruction also. Then he would go along perfectly well until the plum pudding started to move again, when he would have colic and obstruction. This state of affairs continued until the mass finally came to the smallest portion of the intestinal tract, which is about 39 inches above the ileocecal valve. There it stopped and produced a typical intestinal obstruction. We removed the plum pudding.

That shows exactly how these conditions occur. I mention that case to accentuate the absence of colic while the foreign body is arrested in its transit: while it is in a diverticulum. At one time I made the statement, in error, in Sajous' Medicine, that when a patient had a stone in the common duct there was always more or less bronzing of the skin. That is not true. If the stone is an angular stone, the bile goes by without any trouble at all and there will not be any jaundice. If it is the ordinary round stone, there are jaundice and bronzing. In other words, the common duct must be obstructed completely before jaundice can occur. There is no evidence that this patient before us ever had a stone that went down into the common duct. Why? Because she never had a jaundice in any of her attacks.

There is one condition that resembles that, and that is the recurrent attacks of colic from gastric ulcer. I recall the first case of that kind I had. The patient was a police officer. When he told me his story I felt that he must have an occlusion of his gall-bladder. His history resembled that of a gall-bladder lesion. I saw him while he was having an attack. It was clearly demonstrated that it was the recurrent colic of a gastric ulcer. We could feel the ulcer high up. We operated and showed that that was the cause of the recurrent colics.

We will now expose the gall-bladder through the usual incision over the right rectus muscle. In the examination of this woman the liver seemed high up under the costal arch, and inspection confirms that finding. She has a dilatation of the duodenum,

not enough to call it a megaduodenum, but still a larger duodenum than normal. The pylorus is perfectly patent. There is no evidence of recent or remote ulceration here. This zone is free from adhesions. I will now displace the stomach and the intestines out of the field to bring into view the gall-bladder. I do not see the gall-bladder. It does not come into the field in the usual way. The liver is in the field, but the gall-bladder is not. As I move this down, I confirm the observation made before the operation, namely, that the gall-bladder is higher up than usual, namely, that it is not in range. I must endeavor to get a little more opening here. The liver comes down, but the gall-bladder does not come into the field. But what does come into the field? A thick adhesion at the position where the gall-bladder should be. I do not see the gall-bladder, but I take it that this is the result of a condition not mentioned in the history and not indicated by the history. Did she ever have an inflammatory attack that confined her to her bed for any considerable period of time? That is a question which is suggested by the findings here. We must separate these adhesions from the liver and endeavor to find the gall-bladder in there somewhere.

Before we do so, however, let us look again because these conditions are not in consonance with the history. Ah! there is the gall-bladder to the outer side of these adhesions and hanging away down under the edge of the liver. It does not look like a gall-bladder at all. It looks like the large intestine in color, size, and shape. It is red from the continued inflammatory condition, and elongated and dilated. It is at least four inches in length. There are some masses down in its neck. Whether they are gall-stones or enlarged glands around the neck I do not know. I take it they are gall-stones down in the cystic duct, producing a chronic retention of bile in the gall-bladder.

There is a perfect pathologic record here of the pericholecystitis that had been present at different times. There is nothing in that story to tell us whether any of the attacks were severe enough to confine her to bed for a long time after an attack, or whether she ever had a temperature elevation with an attack, but these adhesions show how severe that inflammatory process

was. The adhesions are old and well organized. They are the sequence of a severe infection.

We will take this gall-bladder out. Why? Because to leave it in means to invite a return of the trouble. Here are some foreign bodies, which are gall-stones, embedded in the cystic duct. I am able to luxate them back into the gall-bladder. I will now free these adhesions around the neck of the gall-bladder so that the cystic duct will be accessible. What about the urinary findings in this case?

INTERN: The urine is negative.

DR. MURPHY: That is all right. There is the cystic duct. We clamp the neck of the gall-bladder with the rectangular gall-bladder forceps to prevent the stones and secretions from flowing out when we remove the gall-bladder and contaminate the field. We will ligate and divide this duct between two ligatures. Let us examine the common duct. I pass my finger inside this zone of adhesions and on down into the zone of the common duct. There is no stone in that duct within the range of my finger.

Let the record show that there is no thickening of the head of the pancreas, showing that the infection that has existed here has not materially involved the head of the pancreas.

I want to bring this tissue all up into the field so that I can make a better examination of that common duct. We will free the adhesions and that will come right into the field. I will make a careful analysis of the entire situation. This is just the type of case in which one could leave something that would give the patient annoyance later. That rectangular forceps on the gall-bladder does not injure the gall-bladder. The doctor holds it up in position while I make a careful examination of this duct. That is the forceps put on the gall-bladder at the cystic duct to prevent the stones slipping back into the cystic duct. I have it all in my field. There is the hepatic artery. There is still a stone in the cystic duct. Let me see if I have them all out. Yes; this single stone slipped back into the cystic duct after I got all the stones through into the gall-bladder. There are no stones in the common duct. Now we will amputate the gall-bladder, proceeding as we did in the previous case, and a drainage-

tube is secured to the stump of the cystic duct with the same ligature that we used to ligate the duct. All the bile that may come out through the stump of the cystic duct must pass on out through that tube. The tube is fixed to the skin with a silk-worm-gut fixation stitch. The abdominal wound will be closed in the usual manner.

Let the record show that the gall-bladder was very materially enlarged, extending about three inches below the margin of the liver; that there were small stones in the cystic duct in large numbers; that evidently there had been a severe inflammatory condition, because all the structures in this neighborhood had become organically adherent to the gall-bladder.

The history of this case does not fit this pathologic condition. There is no record of a pericholecystitis and no record of a peritonitis in the upper abdomen. That record must be there. It is there somewhere; there is no question about it. She could not have these adhesions to such an extent without having had some severe attack. There are, you see [examining specimen], a large number of gall-stones of all sizes and shapes in the gall-bladder. The wall of the gall-bladder is thick and infiltrated.

Let the record further show that all the stones in the cystic duct could be pressed back into the gall-bladder; that we clamped the cystic duct and amputated it, and touched the stump with iodine; then we fixed to that ligature a drainage-tube, so that the drainage-tube remains over the stump of the cystic duct.

If the ligature gives and the bile leaks, it will come out through that tube as the easiest means of getting to the surface. Agglutination will take place around the tube after eight or ten hours. Long before the pressure increases enough on that gall-bladder the rubber tube will be surrounded by a connective-tissue tube, so that it is perfectly safe to treat it in that way. Let the record also show that there was no ulcer of the stomach or duodenum, and that the duodenum was a little dilated.

This woman had a history of colic extending over a period of five years and without ever having had a jaundice. In our analyses and statistics of jaundice in relation to cholecystic colic we found in the cases we had operated on that 86 per cent. never

had jaundice as a symptom; therefore, jaundice is only one of the late symptoms in many of the cases and never necessary in order to make a diagnosis. The clinical course, the ordinary physical examination, which you can make, without the presence of jaundice, is all you need to make a diagnosis. The jaundice never comes until the stone goes down into the common duct. We should put jaundice out of the syndrome that is essential in making the diagnosis.

We had a patient who had these attacks for thirty-one years, and she never had a jaundice. She had something like 400 gallstones in her gall-bladder. The one in advance was a large stone down in the cystic duct. During all that time she was treated for gastralgia, which was relieved by vomiting, as these cases are practically always, and therefore are called "stomach trouble."

In the management of these cases we are all coming to one consideration, that the time to operate is when the patient has the original attack. That is the time. You should not have to wait for a superlative pathology to develop, any more than you have to wait for an abscess to form in the abdomen in connection with appendicitis. *Operate in the initial attack!* We must educate the people to the necessity of operating early in cholelithiasis, the same as in appendicitis. You cannot hold an attack of appendicitis, even with ordinary people, for more than twenty-four hours. If you do, and something happens, you get what you deserve from the people. That is the nice part of it, that the people work with us if we are positive in our convictions. It is a fact that they can see that we are wavering even better than we can. You can take an educated fellow and he will believe a plausible story, but the common people—the uneducated people—have the "horse-sense" that counts. That has always been true.

Let the record show that Sister reports the sponges counted and correct.

ACUTE PANCREATIC CYST

With Discussion of Medical Phase by Dr.
Charles L. Mix

DR. MURPHY (Operation, November 15, 1913): This case is a very interesting one from a diagnostic standpoint. I had hoped that Mr. Paterson would operate on this patient, but it is not fair to give this kind of a case to a visitor. However, I will ask Mr. Paterson to talk to us about the surgery of the duodenum and stomach while I go on with this operation. Dr. Mix will take up the medical phase of the case.

HISTORY

Patient, male, aged fifty, entered the hospital November 14, 1913, because of a mass in the epigastric region. It is of about the size of a man's fist, rather firm, somewhat tender to touch, and painful. It is visible and can be felt pulsating. He also has a reducible left inguinal hernia, not tender or painful, and giving a decided impulse on coughing. The hernia was first noticed eight years ago, since which time it has gradually increased in size, but never has been painful or tender. It becomes larger when he is constipated. His bowels are constipated, and have been so for the last sixteen years.

The patient states that he was perfectly well until three weeks ago, when, while working one night, he developed a severe, cramp-like pain in the epigastric region. He did not vomit, but belched a great deal. He went to a drug-store and the druggist gave him something which relieved the pain. The next day he felt well, except that from one to one and one-half hours after meals he would have pain in the epigastric region, which was followed by belching. He did not become nauseated nor did he vomit.

He then began to feel tired, weak, and sleepy. He consulted a doctor, who, without examining him, said he had chronic dyspepsia. The doctor gave him some medicine, which relieved the pain, but he had a constant soreness in the epigastric region, and on pressing over the pit of his stomach he noticed that this region was somewhat tender. On the third day he discovered a hard mass in the epigastrium which was tender to touch. His appetite seemed to be good, and he ate as well as usual, but he felt tired, weak, and sluggish.

He consulted another doctor, who said he had "a lump in the membranes of the stomach." He, too, gave him medicine, which he said would reduce the size of the lump. Since this time he has been eating about as well as usual, but he has had a constant soreness in the epigastric region. He thinks the tumor has diminished in size since he first noticed it.

He has lost 15 pounds in weight in the last year. His bowels have been constipated very much during this time, so that he has had to take cathartics continually. He has never noticed any blood in the stools nor has he vomited blood. He denies ever having had jaundice, a venereal infection, or lues. There is no history of tuberculosis or carcinoma in the family.

A test-meal was given early this morning. The examination showed a total acidity of 80; no free hydrochloric acid; lactic acid strongly positive, and a trace of occult blood. The blood count is 13,900 leukocytes and 3,328,000 erythrocytes, with a hemoglobin percentage of 70. The urine is negative for albumin, sugar, and casts. The specific gravity is 1032, reaction, acid.

DR. MIX: You have just heard the history of the case and will note the statement that the patient denied venereal infection and that there was no history of lues. The first noticeable physical sign in looking at the patient is the pupillary inequality, which immediately led to the discovery of a specific infection. The right pupil is larger than the left, and it is irregular in outline. This inequality and irregularity combined are safe evidences of an existing syphilis. The inequality by itself has no invidious meaning. The inequality with the irregularity of the pupil is an excessively strong evidence in favor of a syphilitic

infection. This infection, I learned on questioning the patient, took place when he was twenty-three years of age.

He is a police officer. His weight a few years ago was 247 pounds. He reduced himself by dieting, in order to be able to get to be a police officer, to 202 pounds. After receiving his appointment he gained weight, going up to 225 pounds, but subsequently again reduced himself to 202 pounds. In the last year he has lost 15 pounds.

He has always considered himself a well man until recently. The onset of his trouble was on the nineteenth of October, when he was seized with a severe, cramp-like pain in the epigastrium, not associated with vomiting, which he thought was due to indigestion. This pain was a continuous pain, and it was not affected by the ingestion of food. I saw the patient for the first time last night at 8.30, so that I have had to complete all the work of examination in a short space of time.

Reverting to the history, we find that on October 20th his appetite was good, and that he ate his meals, but that from one to one and one-half hours after eating them he was troubled with belching. He was not nauseated and did not vomit. He apparently had some infection, because he complained of having been tired, weak, and sleepy. He saw a physician on the twentieth who, without examining him, told him that he had chronic dyspepsia and gave him a prescription. This prescription relieved his pain, but the soreness in the epigastrium remained. During the next six days he remained in practically the same state as he was when he saw the physician, but on October 26th he had a very severe pain in his epigastrium, which was tender to touch. He, therefore, visited another physician, who examined him and found a lump, as he expresses it, in the membranes of the stomach.

In the history mention is made of two tumors. That which occurred in the left inguinal region has already been explained by Dr. Murphy as being due to a hernia. The other tumor, which is about the size of one's fist, or perhaps somewhat smaller, is situated in the epigastrium. It is rather firm in consistence and has a reasonably sharp outline. In this latter respect it does not

feel like an ordinary granuloma. In infiltrating tumors or tumors which are surrounded by an area of inflammation you will find a tumor mass with a vague outline, shading off into the other tissues and therefore having more the feeling of an irregular lump than that of a sharply defined tumor. You may decide at once by the "feel" of the tumor mass whether it has a distinct capsule or whether it consists of an ulcerated mass of some sort shading off into the surrounding tissue, which it mats together in a mass in the abdominal cavity. In the case of this tumor the edge is sufficiently sharply defined to enable one to conclude that it is probably not surrounded by a very large inflammatory area.

A second interesting fact concerning this tumor is that it does not move with respiration. This might indicate many things, but, as a rule, a fixed tumor in the abdominal cavity is one which is fixed to the posterior abdominal wall. A fixed tumor raises the presumption of a retroperitoneal sarcoma, of infiltrated and enlarged mesenteric glands, of a tumor of the kidney or pancreas, or possibly of an aneurysm of the abdominal aorta. A movable mass in the abdomen raises the presumption of a tumor involving either the liver, stomach, spleen, or intestine. Not all kidney tumors are fixed to the posterior wall—many of them are movable. From the fact that this tumor does not move with respiration we feel that it must be a tumor of the sort which is fixed to the posterior abdominal wall.

Another important observation concerning this tumor is its location. Apparently it rests sufficiently far forward to lie close beneath the anterior abdominal wall. Its position in the epigastrium is such that it becomes necessary to determine as accurately as we can its relation to the liver and pylorus. I find that it is possible to place two fingers between the tumor and the edge of the liver, and to get a perfectly good percussion-note in this small space. To me this is positive proof that the tumor is not connected with the liver. If it were so connected, two things would happen: First, it would move with respiration; and, second, no clear percussion-note could be obtained between the two masses.

The relation between the tumor and the pylorus was not

difficult to demonstrate. On passing the stomach-tube this morning and inflating the stomach with the Ewald bulb I was able to demonstrate satisfactorily to myself that the tumor mass lay near the stomach, but that it was not a part of it. Thus far, then, we may conclude that the tumor mass with which we are dealing is not attached to the liver; that it is not a part of the stomach and pylorus; that it is undoubtedly attached to the posterior abdominal wall; that its size is sufficient to protrude as far forward as the anterior abdominal wall.

The last point in connection with this tumor was mentioned in the intern's history. You will recall that the intern said that the patient, a male, fifty years of age, entered the hospital because of a tumor mass in the epigastric region, about the size of a fist, rather firm, and somewhat tender and painful, and which could be seen and felt pulsating. If the intern's observation is correct, the tumor is very clearly an aneurysm of the abdominal aorta. The feeling of the edge of the tumor is, however, not sufficiently clear cut for an aneurysm. Moreover, the intern neglected to differentiate between a tumor which apparently pulsates and a tumor which is expansile. I am sure if the intern had carefully examined into the matter from this point of view he would have concluded that the tumor was not an expansile tumor, and that pulsation felt was communicated to it by the abdominal aorta.

In favor of the conclusion that this tumor is an aneurysm, notwithstanding the absence of a sharp outline and the absence of the expansile symptom, it will be found, on listening with the stethoscope, that a systolic murmur is present. This murmur seems very much like an ordinary systolic murmur of any aneurysm. Feeling the pulsation, and hearing this perfectly apparent murmur, you would be almost certain, without some analytic care, that the tumor is an aneurysm. If, however, you turn the patient upon his face and listen to the abdomen while he is bent on his elbows and knees, you will find, as I found when I made the experiment last evening, that the murmur will disappear. In other words, that murmur which is heard is caused by the compression of the abdominal aorta by the tumor mass. You

can get a murmur exactly similar to the one heard in this abdomen by compressing very firmly the femoral artery in any individual and listening with the stethoscope. We, therefore, must conclude, since the tumor mass is not expansile, since the murmur, which it apparently has within it, lies behind it, since its edge is not the edge of an aneurysm in outline, that the diagnosis of aneurysm is untenable in this patient.

Is the tumor mass a carcinoma of the stomach? An ordinary carcinoma of the pyloric end of the stomach can be moved about to some extent by the hands of the examiner. It is seldom true that it is so firmly anchored as to be perfectly immobile. This particular tumor cannot be rolled about in the abdominal cavity, either by the patient taking a deep breath or by the examiner pushing it with his hand.

To conclude as to whether it is a carcinoma, we must necessarily inquire into our laboratory findings. We have had so little time to go over these that we could not resort to all the laboratory work that we usually do in these cases. The laboratory results are very useful, particularly with regard to occult blood in the feces and in the stomach-contents. We have had the patient's feces examined, and occult blood was found in them in large amounts. In the stomach test-meal given to the patient last night at about nine o'clock we found occult blood, not reported in the previous Ewald meal which he had taken. Lactic acid was also found in the stomach-contents taken after this 9 P. M. test-meal. Furthermore, we found that it contained no free hydrochloric acid and the combined acid was rather low. This morning, previous to inflating his stomach to determine its relation to the tumor, I removed with the stomach-tube the contents of this glass (showing glass containing about four ounces). He said he had not eaten anything since yesterday, and yet we got this amount out this morning. We have not had time as yet to have this specimen examined in the laboratory, though it looks as though it contained blood acted upon by hydrochloric acid. The contents of this glass indicate that he has some retention, due either to a muscular atony or to some sort of stenosis of the pylorus, presumably the mechanical effect of this

tumor. Whether the residuum which we found means muscular atony or mechanical stenosis is, of course, difficult to decide.

Putting all these facts together in answering the question as to whether the tumor is a carcinoma, we find them to summarize as follows: First: There is some sort of pyloric block; second: there is no hydrochloric acid; third: lactic acid is present; fourth: occult blood is present in the stomach-contents; fifth: occult blood is present in the feces; sixth: there is a tumor mass which is situated in the epigastrium, hard and firm in consistence, and not perfectly sharply outlined. Such an assembly of facts would ordinarily be regarded as almost positive proof of the presence of a cancer of the stomach. Against this array of facts, however, we must place the statement, which I have already made, that on inflating the stomach the tumor was found not to be a part of it; further, that no Oppler-Boas bacilli were found; further, that the tumor mass cannot be rolled about in the abdominal cavity: that it is apparently firmly fixed and moves neither with respiration nor with the examiner's aid. It is obvious that the diagnosis of carcinoma of the stomach is open to very serious question.

There is another point in the history of this case not mentioned by the intern, which to me is exceedingly significant. When I saw him this morning I asked him what he ate yesterday, and among other things he said pie. A man who has carcinoma of the stomach is not in the habit of eating apple-pie. A man with a late carcinoma of the stomach would not have the nerve to eat apple-pie.

Again let us revert to the history. It must have occurred to you on hearing it as the intern read it that this patient's discovery of the tumor was rather sudden. The tumor must have developed in an amazingly short space of time, according to his own statement and that of his wife. The man was in comparatively good health previous to October 19th. On October 26th the tumor was present, and sufficiently large for the patient to be able to discover it himself. The first physician who looked at him found no tumor, but it is true that he prescribed for him without removing his clothing. On the twenty-sixth of October

this mass had attained a size so great that anybody could feel it. Either one of two things is true—either the patient's observation was exceedingly careless or the tumor was a very rapidly growing one.

Such a rapidly growing tumor may result from the pancreas. A sudden closure of the duct of Wirsung or ranula of the duct will cause the development of just such a tumor mass, and cause the sudden development of a pancreatic cyst. This sudden discovery of the tumor on the part of the patient is, therefore, very suggestive of a pancreatic cyst, rather than of a carcinoma of the stomach. Ought such a sudden closure of the duct of Wirsung to lead to a jaundice? By no means, unless the common duct is also blockaded.

The short space of time in which this tumor developed; its position in the epigastrium, and its demonstrable freedom from attachment to both the liver and pylorus; the fact that it is fixed and does not move on respiration, that it has no anatomic relationship with the abdominal aorta although he has a pronounced pulsation communicated to it, that it lies between the stomach and the colon as nearly as I was able to determine, that it is associated with occult blood in the stomach and in the feces—are all points strongly presumptive of the diagnosis of pancreatic cyst. The location is exactly right for a pancreatic cyst. Pancreatic cysts usually appear between the stomach and the colon. Unfortunately, I did not have the opportunity to inflate the colon in this patient's case, because he had to be operated upon this morning, and it would be obviously wrong to have the colon distended just before the operation. I am, however, certain that the colon was below the tumor, which lay between it and the stomach above.

Did this tumor result from an acute pancreatitis with the sudden formation of a pancreatic cyst, or is his case an old chronic pancreatitis with a cyst? Inasmuch as there is no history of a previous disturbance, it is not likely that chronic pancreatitis was formerly present, though it may have been present without any symptomatology. A chronic pancreatitis usually does not produce the severe symptoms which this patient had. *An acute*

pancreatitis, on the other hand, can cause blood to appear in the stomach-contents and in the feces. A rather mild case of acute pancreatitis, if we were able to diagnose it, would probably be found to present a set of symptoms not very much unlike those of dyspepsia, associated with pain, not relieved by the ingestion of food, more or less continuous, not very severe, but rather a dull aching pain, with blood in the stomach-contents and the feces. In a general sort of way these are pretty much the symptoms which the patient had previous to the discovery of the tumor mass.

I have been asked if I made use of the Cammidge test. No, because it cannot be done in a few hours' time. You know, furthermore, that the reaction does not always turn out positive; that is, the fat decomposition products do not necessarily appear in the urine in cases of pancreatic cyst. I cannot say whether the Cammidge reaction was present or not.

A final word must be said in regard to the possibility of a syphilitic etiology in this case. Gummata of the pancreas are exceedingly rare. I have never seen a case and I do not believe that ten cases exist in the literature. I do not see how a syphilitic lesion could develop a tumor in so short a space of time as I believe it to have been developed in this case. I am, therefore, of the opinion that the luetic infection does not figure in this case.

Our final conclusion is, then, that the tumor in the upper part of the abdomen is probably a pancreatic cyst.

DR. MURPHY: That is a very good presentation of the case. The history lacks some of the elements that are commonly associated with acute pancreatitis, such as vomiting and collapse, nor do we know whether there was any elevation of temperature. The most severe type of pancreatitis is the acute hemorrhagic type, which is initiated by the most severe colicky pains in the epigastrium. As Dr. Mix said, it resembles closely the pain of perforation of the stomach, but it is much more severe. The absence of generalized abdominal rigidity and the early onset of meteorism are points against perforation of the stomach. I recall the case of a man who, while walking on the street, had such a severe attack of pain that he had to sit down on the near-

est door-step. He was unable to stir and would not permit himself to be removed until the police patrol arrived, when he was forcibly removed to the hospital. He screamed every time any one came near him. I merely mention that case to give you an idea of the severity of the pain in the acute type of pancreatitis.

This case today is of the less acute type. This man came home at midnight, had pain in his epigastrium all night, and in the morning he consulted a physician. This is not the history of a carcinoma of the stomach. It does not come on in that stormy fashion. The temperature in this case after admission to the hospital was 99.6° F. in the afternoon and 100° F. later in the night. The leukocyte count was 13,900. Remember this attack began three weeks ago, on October 19th, so the present temperature record has not at all the significance that a temperature record in the early days of the attack would have had.

The onset, with pain and epigastric tenderness, is fairly distinctive in this case. Remember that the etiology of this condition is still obscure. An ascending infection from the intestinal tract has been primarily the essential factor. A metastatic infection might occur, but is rather uncommon. It is believed by many that the duodenal contents regurgitate into the pancreatic duct in certain conditions, and that this locks up the pancreatic ferment, which in turn causes a digestion of the gland. This may be followed by a bacterial invasion. This digestion of the gland affects the blood-vessels, leading to hemorrhage in and around the gland. The process then spreads beyond the confines of the gland, leading to fat necrosis. It may also be a sequence of an infection in the gall-bladder involving the common duct and the duct of Wirsung.

We are now ready to make the incision in the epigastric region, going through the right rectus muscle-sheath. On opening the peritoneal cavity we find much edema of the tissues, and on exposing the pancreas see presenting in the field a granular-looking mass with many minute, opaque white areas scattered through the omentum and mesentery—areas of fat necrosis. It is an acute condition from the pancreas. The trypsin fat-splitting effect shows clearly here. The mass presses well up

to the anterior abdominal wall. Shall we incise this mass? No. We shall just put in a coffer-dam of nosophen gauze, shutting off the peritoneal cavity, and in five or six days we shall remove the gauze and open into that abscess in the pancreas. We shall close the abdomen in the usual way, and from the upper angle of the wound the end of the strip of nosophen gauze will protrude.

Let the record show that this was an acute pancreatitis; that the inflammatory mass had not become adherent to the anterior abdominal wall; that a coffer-dam of nosophen gauze was put in; that we will wait for five or six days before opening the abscess.

Let the record show that Sister reports the sponges counted and correct.

[We subsequently opened the abscess and drained it. The tube was taken out after three weeks, the abscess refilled, and it had to be reopened.]

Further Report on This Case, March 1, 1914, by Dr. Charles Louis Mix.—Following the original operation, the pancreatic cyst, which had a rather thick wall, discharged for some weeks. Unfortunately, however, vomiting started in with a good deal of persistence, indicating serious disturbance either by reason of a reflex or a direct involvement of the stomach. The latter proved to be the actual condition, because through the drainage-tube an acid secretion appeared which on analysis proved to be gastric juice. Inasmuch as the opening into the more or less indurated cyst showed a tendency to close, the drainage into the stomach being apparently the method of drainage which nature insisted upon, and inasmuch as the prognosis thereby became excessively grave, it was thought wise to permit the drainage opening to close, which it did. Subsequently a nodule formed in the costal border of the liver, which was plainly carcinomatous. For that reason the original pancreatic tumor must be looked upon as carcinomatous, either from the beginning or becoming so at some time subsequent to the operation. The metastasis in the liver and the continuous discharge from the tumor mass into the stomach led to the gradual wearing out of the patient, and he

ultimately died from exhaustion and a worn-out myocardium. No postmortem examination was made.

DR. MURPHY: Let me show you a few operative cases that accentuate some very valuable points. The first is a case of posterior gastro-enterostomy, done for a gastric ulcer. We do the posterior gastro-enterostomy with the short loop. We usually use the oblong button for making the anastomosis. The operation is done quickly, and when it is technically correct, there is absolutely no danger of hemorrhage or leakage. The reports coming from the European clinics where the button is used more or less extensively show that it prevents post-operative hemorrhage. (See April, 1913, CLINICS, p. 219.) The greatest mortality from gastro-enterostomy now is due to pneumonia; next comes hemorrhage, which the button always controls. We have not had a single unpleasant result from the use of the oblong button. This patient passed the button on the fifteenth day.

Here [indicating another patient] is a most interesting case. I want to speak of this case because it teaches a valuable lesson.

This patient was taken severely ill on Thursday, October 9th, with a severe, cramp-like pain in the epigastrium. She came here on Sunday, October 12th. She had been vomiting almost constantly since Thursday. She had abdominal discomfort the early part of the week, but did not get ill until luncheon time on Thursday. A doctor was called, and *he administered a hypodermic of morphin to give her relief from the pain.* That is the point I want to emphasize. Pain means what? Pain is an announcement that there is something wrong; that there is some pathology somewhere in the patient's body, and it is the physician's duty to interpret the pain. Don't mask the symptoms by giving morphin or any other opiate. Pain is a danger-signal, and it is our duty to recognize it as such. Ascertain why the patient has pain. That is the proposition.

A few days ago a patient was brought to the hospital with a perforated duodenal ulcer. He was taken ill early Saturday morning. He called in a doctor at once, and the doctor gave him one hypodermic of morphin. That did not stop the pain, so he gave him another and another, until the patient was com-

fortable and the doctor was secure, because he could not have held that case without making a diagnosis if the patient continued to scream with pain. But he failed to make a diagnosis. He did not discover the cause of the severe pain. The patient came to the hospital on Monday night. My associates made a diagnosis on the basis of the man's story. They operated at once, but more than forty-eight hours had elapsed since the perforation occurred. A perforated duodenal ulcer was found, but the man died. The morphin had given him relief from his pain, but the pathologic process continued active and that led to his death. That is the proposition. That is the point I want to bring out.

To return to the preceding case. There is another element in this history which is instructive, and that is, that she had an enema and got a copious bowel movement afterward, although she had been given morphin about three times a day for two days to give her relief from pain. She had had no natural bowel movement since the onset of her illness. She gave a typical history of an acute intestinal obstruction. The fact that she got a copious discharge of feces from that enema was no indication at all that the intestine was not obstructed. She had a typical ileus, as you see ileus mapped out on one of our charts hanging on the wall.

On opening the abdomen we found the ileum obstructed by a band of adhesions extending from the anterior abdominal wall to the brim of the pelvis. The patient had been operated on before for some tubal trouble, and she had had a tubercular peritonitis which we had treated with tuberculin and from which she has entirely recovered, as was shown by the examination of her peritoneum at the time of the operation. Her tuberculin reaction is negative. What do you do when you open the abdomen in a case like this? First get the distended coil of intestine. That is always in the field. Then locate the ileocecal junction, and pass the ileum quickly through your fingers, returning the coils of intestine into the abdomen as rapidly as they are passed out until the point of strangulation is reached. Then free the intestine by dividing and ligating the constricting band,

and, if necessary, make a resection, or, if not, and the intestine shows that its circulation has not been seriously damaged, cover it over with warm towels until little by little the circulation is returned. Then return this loop of intestine back into the abdomen and close the wound. Always wash out the stomach thoroughly in these cases before the operation, because otherwise the patient may get a toxemia. We keep most of these patients in a semi-sitting position during the operation. Why? Because that keeps the stomach-contents from flowing back through the esophagus and into the lung, exciting an inhalation pneumonia, which is usually fatal in these cases.

We used nitrous oxid gas anesthesia in this case throughout the entire operation, as we always do in these cases.

The mortality of intestinal obstruction has not materially diminished in thirty years, notwithstanding all our improved technic in handling the condition. Why? Because the patients are practically dead before they are operated on. That is the proposition. That is why we should give heed to the symptoms, and, remember, in the diagnosis of intestinal obstruction, the importance and significance of pain, nausea, and vomiting.

This patient has recovered entirely. She is ready to go home.

DUODENAL ULCER; EXTENSIVE ADHESIONS; DILATED STOMACH; GASTRO-ENTEROS- TOMY. DESCRIPTION OF DR. MURPHY'S BUTTON OPERATION

Discussion of Case by Dr. C. L. Mix

HISTORY

PATIENT, male, aged twenty-one, entered the hospital November 5, 1913, because of intermittent attacks of burning pain and fulness in the epigastric region, coming on from one to two hours after meals, causing him to belch, and, in the course of time, to vomit.

The present trouble dates back to 1909, when he first noticed a sense of fulness and a heavy feeling in the epigastric region, coming on about two hours after eating. He consulted a doctor, who told him he had indigestion and treated him for this. These attacks continued off and on until 1910, when the patient began to have pain and a gnawing feeling in the epigastric region, and he vomited. The vomitus was sour, and at times contained dark blood. He again consulted a doctor, who put him on a liquid albuminous diet for six weeks. It seemed to relieve the trouble. He again began to take a general diet and the symptoms returned.

From this time until May, 1911, he had intermittent attacks of pain and vomiting, and each time he would be relieved by returning to a liquid albuminous diet. The return to a general diet was followed by a return of his symptoms.

May 6, 1911, while riding in a wagon, he was thrown out, striking the ground with head and right shoulder. Following this he had severe pains in the back, at the level of the lower dorsal and upper lumbar vertebræ, which continued for fifteen minutes and then ceased. About one week following this injury he was awakened at 6 A. M. by a sharp pain in the epigastric re-

gion, which soon became generalized. He vomited, but does not remember what the vomitus contained. A doctor was called, who gave him morphin to control the pain. He soon became delirious.

At 7.30 A. M. he was taken by train to a hospital, and at 7 P. M. the same day he was operated on. The appendix was believed to be the cause of the trouble, and it was removed. Two drains were inserted. After the operation he was told that he had a perforation of the duodenum. The first four or five days following the operation the patient was very sick. On the seventh day one tube was removed. On the tenth day he developed a high fever, became delirious, and the tube was again inserted. He was told he had a retention of pus. After one week one tube was again removed, and the other one was removed the following week. He left the hospital after a month with the wound apparently healed. After this he got along fairly well, gaining in weight and strength.

August 3, 1911, he again began to have the fulness and pain in the epigastric region, coming on between one and two hours after meals, causing him to belch considerably. This would be relieved by vomiting, which occurred in from fifteen minutes to three hours after the onset of the pain. The vomitus was sour and often contained bile and occasionally dark blood. His physician put him on a diet of cream, milk, soups, beef, and chicken, but still he was not relieved.

Since this time he has had intermittent attacks of the same nature. These attacks occur from one week to two months apart, and persist from a day to a week. When these attacks came on the patient usually starved himself, because food seemed to aggravate the trouble. From April to June, 1912, he was in the care of a doctor, who gave him medicine which seemed to relieve his trouble. He had no attacks during this period. Since that time, however, he has had a recurrence of these attacks. He has not lost weight nor has he noticed any blood in the stools. The urine is negative.

COMMENTS AND OPERATION

DR. MURPHY: You have heard the history. Dr. Mix, who has charge of the medical work in our borderline cases, will present the case from a diagnostic standpoint.

DR. MIX: The history which you have just heard is a very excellent history, with one exception. The exception is that the sequence of events in the development of the patient's case has not been carefully brought out. I saw this man last evening and mentally made a diagnosis of duodenal ulcer while hearing his story and before I had learned of his operation.

The trouble first began when he was eighteen years of age—that is, three years ago. The first thing he noticed was a sense of fulness in the epigastrium or in the upper part of his abdomen in the midline, which fulness came on at definite times, that is, about an hour or two after his meals. This fulness was relieved in one or two hours, either spontaneously or by the taking of additional food. These two facts—the time of onset of the fulness and its relief by the taking of additional food—are sufficient to *suggest* a diagnosis of duodenal ulcer.

Now the next step in the development of his case was three months later. In addition to the fulness after meals at this time two more symptoms appeared, one of which was vomiting and the other of which was pain. These two symptoms came on together. That is an important point. They are associated, the cause producing the pain being the same cause that produced the vomiting. The epigastric pain and the associated vomiting refer us at once to the gall-bladder, stomach, duodenum, or possibly the pancreas, and to the peritoneum covering these organs.

Another interesting point in regard to the time of the onset of this disturbance is that it was apparently associated with the seasons. It is characteristic of duodenal disturbances that they come on fairly regularly in the fall of the year. If the duodenal ulcer is, as I believe it to be, an infection of some sort leading probably to a thrombosis of one of the arteries given off by the main trunk of the gastroduodenal (which you will remember supplies both the gastric and duodenal side of the pylorus, it

being the artery which is immediately involved in duodenal and gastric ulcer), you can see that perhaps an infection may have something to do with the seasons. In the spring and fall it would be a simple matter in a condition of bacteremia for the infectious organisms to be swept into this artery, and therefore to give rise to a thrombosis and to the development of a peptic ulcer. At any rate duodenal ulcers have exacerbations in the spring and fall, suggesting some sort of infection.

The onset of the patient's trouble, according to the intern's history, dates back to the fall of 1909. As I have already stated, within three months' time, in the beginning of 1910, the patient experienced his first pain and vomiting. With a treatment consisting chiefly of an albuminous diet, mostly liquid, he got along very well until May, 1911, when suddenly a crisis occurred in his life-history. On May 6, 1911, he told me that while riding in a wagon he was thrown off and severely hurt. It was raining at the time, and in his disabled condition he became thoroughly chilled. Following this fall and exposure he had severe pains in the back, at the level of the lower dorsal and upper lumbar segments. These pains continued for only a few minutes after his fall and then disappeared, and never recurred. He seemed to be in his usual health, when suddenly, about one week after his fall, he was awakened at 6 o'clock in the morning by a very sharp pain in his epigastrium. This pain was associated with vomiting. A severe pain coming on at this time of the day while the patient is in bed, associated with vomiting, is always ominous. A physician was called, who gave him morphin for the relief of the pain, and by so doing completely muddled up the symptoms. If there is any one thing which must be sharply blamed in the management of these cases, it is giving morphin to a patient with such a set of symptoms. Many a person has lost his life by the undue haste of a physician to lodge a quarter of a grain of morphin beneath his skin, obliterating the symptoms and clouding if not preventing the diagnosis. At 7.30 A. M. the patient was very properly rushed off to Milwaukee and operated on that night at 7 o'clock.

It is perfectly obvious from the history which has been given

of pain coming on at a definite time after the ingestion of food, of pain relieved by the further ingestion of food, of pain relieved also by vomiting, and, lastly, the vomiting of dark blood, that a duodenal ulcer was *then* present. We are told that a diagnosis of appendicitis with a ruptured appendix was made. It must be perfectly obvious, from what I have already said, that no one point in his history speaks for the presence of an appendicitis; whereas the history is, in the time of year of onset, in its course, in the relationship of the pain and the time of eating, in the relationship of the relief of the pain with the further taking of food, and the vomiting of blood, a perfect one of duodenal ulcer. It was at this point, before I heard of the operation, while listening to his story of what occurred early in May, 1911, that I made the mental diagnosis of a perforation from a duodenal ulcer. I was not surprised to hear that a diagnosis of appendicitis had been made, because it is the one usually made on such occasions, although a perforation from a duodenal ulcer is by no means uncommon.

We do not know the specific course of the case after the operation for appendicitis, nor do we know exactly what was done, except that two drainage-tubes were inserted. We do know that after the removal of one of the tubes the patient developed a high fever, that he became delirious, and that it was necessary to insert the tube once more. He was told that there was a pocket of pus in the bottom of the wound. Such a history raises the presumption that he has adhesions in his upper abdomen at the present time.

Despite the temporary relief obtained by the operation early in May and his subsequent gain in weight and in strength you will note that the trouble again broke out early in August, 1911. We find the same old symptomatology once more, that is, fulness and pain in the epigastric region coming on one to two hours after meals and relieved by vomiting. But we also now note a new point in the intern's history, and that is, that in addition to the facts previously stated about the vomitus, namely, that it was sour and occasionally contained dark blood, it now often contained *bile*. Remember that at the beginning the vomitus

was the ordinary watery and slimy vomitus; that within three months' time it began to show blood; that the blood continued off and on up to the time of the perforation, which took place in May, 1911; that the vomiting of blood then ceased until August, 1911; that then bile was also added to the vomitus. The presence of bile means a reflex peristalsis, the bile regurgitating through the pylorus into the stomach and then causing nausea and its rejection. The presence of blood means the advent of an erosion of a blood-vessel in an already existing ulcer. In the history which the patient gave to me he said that the blood sometimes appeared two or three or four days after the onset of one of these spells, indicating that three or four days sufficed for the ulcer to erode deeply enough to involve a blood-vessel.

What is the significance of the appearance of bile in the clinical picture since August 3, 1911? Our observation of a pure case of duodenal ulcer is that the vomiting never takes place unless there be a lesion deep enough to cause either an irritation of the peritoneum or its inflammation. As a rule, the ulcer has to be quite old or else quite deep in order to produce this peritonitic effect. If, however, there is associated with the duodenal ulcer, as is often the case, a secondary gastric ulcer, the vomiting is very common, particularly if the ulcer be close to the pylorus. We also often find vomiting in duodenal ulcer whenever there is an associated pericholecystitis or cholecystitis. If bile is present in the vomitus, it necessarily means a reversal of the ordinary peristaltic wave. To the best of my knowledge and belief the bile cannot pass from the duodenum into the stomach unless in the cases of duodenal ulcer there is an inflammation of the surrounding peritoneum. Such a periduodenitis, perigastritis, or pericholecystitis seems to be competent to produce a reflex antiperistalsis and to carry the bile into the stomach, provided the pylorus is sufficiently patent. When we hear, therefore, that to the symptoms of duodenal ulcer has been added the vomiting of a bile-stained fluid, we must believe that there is some associated peritonitic inflammation. This may seem to be a radical statement, but it is one that has been my personal experience and I thoroughly believe in it. The bearing of this fact

upon this particular case then is that it seems to establish a spread of the inflammation from the duodenum. The fact that a perforation of the duodenum took place is almost of itself a sufficient argument to support the statement that the surrounding peritoneum had been involved, but the vomiting of bile-stained stomach-contents is a proof of such spreading inflammation.

I would like to call your attention to a last point in the patient's history as given by the intern. The intern has noted the fact that after August, 1911, the patient belched a good deal. Such belching may have been due merely to the duodenal ulcer, or it may have been due to the resulting anatomic changes following the duodenal perforation. Belching is extremely common in duodenal ulcer. The patient complains of gas. The primary thing in these cases is the complaint of gas; the belching is secondary, and, as a rule, forced. The patient has a sense of fulness in his stomach which he interprets as the pressure of gas, but which we interpret as due to hyperchlorhydria, present in the majority of cases. In order to relieve himself of the disagreeable sensation he tries to belch, and after making numerous attempts, succeeds in swallowing enough air to be able to get rid of a little of what he has taken in, and then he feels a certain amount of relief. There was no gas in his stomach originally, or if so, but a small amount. As a result of his endeavors, however, he at times acquires quite a good supply of air. To my mind the statement by the patient of a subjective sensation of gas is often equivalent to the statement by the patient of the presence of hyperchlorhydria.

In this history of duodenal ulcer there is one great gap. There has never been a history of black, tarry stools, which, if present, establishes the diagnosis of duodenal ulcer absolutely, provided, of course, that the other characteristic symptoms are present; but we see very many cases of duodenal ulcer with such a history of bloody stools, and we have to make the diagnosis without this valuable aid.

Physical examination of this patient has disclosed, as is usual in cases of duodenal ulcer, but very little. The patient's pains,

which originally were rather diffuse, subsequently localized themselves, and have for some time been situated in what I venture to name the "duodenal area," which is as important in the diagnosis of duodenal ulcer as McBurney's point is in appendicitis. The "duodenal area" is situated a little to the right and above the umbilicus, and deep pressure in the case of a duodenal ulcer will here disclose a soreness. This patient shows a soreness at this point. He also shows soreness over the gall-bladder area. A blow at this point produces distinct pain.

The other symptom which the patient showed on physical examination was the presence of splashing in his stomach long after he has eaten. Splashing in the stomach several hours after meals is a condition of much importance. Though it does not necessarily mean a blockade, it very frequently does. Contrary to the ideas of most physicians, the blockade need not be at the pylorus. Stomach splashing has occurred in several cases of blockade of the duodenum which we have had here in the clinic. We have also met with it when the blockade has been in the first portion of the jejunum. Stomach splashing is to be looked upon, then, as a sign of blockade, either at the pylorus, in the duodenum, or in the beginning of the jejunum. In the case of this patient, on account of the history of an ulcer of the duodenum, together with a history of the vomiting of bile and the history of a perforation, it is likely that a retention of the stomach-contents is due, not to the pyloric obstruction, but to the duodenal adhesions producing a partial block or delay in food-passage.

Examination of the blood showed 3,800,000 reds. This shows that the anemia is not very great, notwithstanding the history given by the patient of the vomiting of large amounts of blood.

We are probably now in a position to come to some conclusion as to the pathologic changes which took place in this man's abdomen from the time of the onset of his illness in the fall of 1909 up to the present time. As we interpret the case, the patient had the formation of a duodenal ulcer. It was duodenal and not gastric, because the pain came two hours after meals, instead of immediately after meals, and because the pain was relieved by eating instead of being made worse by it. The fact that blood

was vomited from the stomach is no indication as to whether the ulcer is duodenal or gastric. This ulcer of the duodenum continued with temporary improvements and exacerbations until May of 1911. Early in this month he was exposed to cold and wet and was thoroughly chilled, and presumably there is some relationship between this exposure and the development of his perforation, which took place early in the morning one week later. Up to this time the case evidently had been a pure case of duodenal ulcer without the evidence of an involvement of the pylorus or of the gall-bladder. After the perforation had taken place, after he had been operated upon and the drainage-tubes removed and a cure apparently established, in August of that same year the trouble again broke out, and now there were added to the picture the vomiting of bile and the retention of the stomach-contents with splashing. There was also added to the physical signs tenderness over the gall-bladder area. It seems almost impossible to escape from the conclusion that following the perforation there was an extension of the inflammation from the peritoneum to the under surface of the liver and to the gall-bladder. There must also have taken place with the healing enough periduodenitis to cause an imperfect drainage of the stomach. We cannot feel that the pylorus has been blocked, because we have no definite signs that an ulcer of the stomach or pylorus ever existed. Had the picture changed as to the time of onset of the pain and the distress after eating, or had the picture changed as to the fact of the relief of the pain by the further ingestion of food, we might assume a new gastric ulcer and a resulting pyloric block, but having no reason for believing in a gastric ulcer, we have not sufficient reasons for believing in the presence of a pyloric blockade.

A duodenal ulcer usually extends its inflammation by way of the lymphatics, spreading to the under surface of the liver and to the gall-bladder. The lymphatics from the duodenum follow the hepatic duodenal ligament to the transverse fissure of the liver. It is, therefore, very common in these cases to note a coexisting pericholecystitis. This manifests itself not by the presence of a jaundice, but by the presence of a tenderness and by

the vomiting not infrequently of a bile-stained fluid. We are compelled to infer that in this particular case such an extension of inflammation has taken place. We, therefore, are of the opinion that this patient is suffering not only from a duodenal ulcer, but also from a certain amount of retention of the food-contents in the stomach by reason of a blockade in the duodenum from resulting adhesions, and that he is also suffering from a pericholecystitis which is of a type sufficient to cause considerable discomfort and reflex antiperistalsis. Mere adhesions between the gall-bladder and the duodenum cause no symptoms by themselves, but if the adhesions be such as to produce traction on the gall-bladder or to produce a constriction of the cystic duct, then the symptoms which they cause are very burdensome and must be relieved. In this particular instance we believe that the adhesions between the gall-bladder and the duodenum are such that there is traction upon the gall-bladder. We do not believe that the cystic duct is in any way constricted, because there is no evidence of enlargement of the gall-bladder. The diagnosis is, therefore, a duodenal ulcer, a periduodenitis with a blockading stricture, and a pericholecystitis.

DR. MURPHY: In order to accentuate the remarks made by Dr. Mix, we have put a special classification of these symptoms on the board, based on an analysis of a large number of cases discussed by a number of men at a recent meeting of the Deutsche Gesellschaft für Chirurgie in Berlin.

Kader in 240 antiperistaltic anastomoses had no case of vicious circle.

Friedenwald in 1000 cases of stomach and duodenal ulcer had 676 in males and 324 in females. Of the 1000 cases, 52 per cent. were duodenal and 40 per cent. gastric.

Seven and eight-tenths per cent. of patients suffering from gastric disorders have ulcer.

Acidity in the 1000 cases:

Normal	Hyperacidity	Subacidity
46 per cent.	30 per cent.	2 per cent.

Acidity in the 52 per cent. of duodenal ulcers:

48 per cent.	35 per cent.	5 per cent.
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Hyperacidity is more frequent in males, and subacidity in females.

The average duration of symptoms is twelve years.

The symptoms are:

Pain	Emesis	Hematemesis	Melena	Occult Blood
94 per cent.	67 per cent.	22 per cent.	51 per cent.	81 per cent.
	54 per cent. (duodenal)		54 per cent. (duodenal)	83 per cent.

A tender area in front in 90 per cent.; dorsal, 32 per cent.

Pain is present in 96.5 per cent. duodenal ulcers; epigastric, 89 per cent.; to the right of the median line in 7 per cent.

Mortality: Petren in 273 gastric and duodenal ulcer cases had 29 deaths.

The postoperative complications were:

Pneumonia, 19 cases, 12 deaths.

Hemorrhage, 3 cases, all fatal.

Peritonitis, 10 cases.

Vicious circle, 13 cases.

Kocher found no carcinomatous degeneration in 80 patients reëxamined one to twenty-two years after operation for ulcer.

Auschultz believes that the frequency of cancerous degeneration following gastric ulcer has been exaggerated. In 930 cases he found but 2 per cent. with cancerous degeneration; of 22 patients on whom resection was made for suspicious ulcer, in only one was cancer found microscopically.

Von Eiselsberg favors Riedel's method of resection, but he did 370 gastro-enterostomies and 50 resections, and only 18 median resections.

The relative frequency of gastric and duodenal ulcer is dependent entirely on the accuracy of the estimate and the exactness of the operator. Originally the relationship of duodenal to gastric ulcer was as 1 to 31 (Greenfeld). While I believe with Mayo and Moynihan that duodenal ulcer is almost as frequent as gastric, this represents the actual situation.

From the modern research work, experimental and clinical, we believe that the etiology of duodenal ulcer is practically always a metastatic infection and not due to hyperacidity. The

sepsis following burns is a simple means of accounting for the duodenal ulcer. Why it should elect this position for embolism and infection is not clear, but that may be said of tuberculosis, osteomyelitis, metastatic arthritis, and many other infections. The experiments of Rosenau and others clearly show that streptococci can produce experimentally both gastric and duodenal ulcers. I believe that there may be a relationship between the infection in the portal tract and the gastric and duodenal ulcer, as appendicitis, ulcerative enteritis, gall-bladder infections, etc., although that is not as acceptable as a theory as the general hematogenous infections. The experimental work of Grundenmann (*Deutsche Gesellsch. f. Chir.*, 1913) is instructive, but not as conclusive as the work of Rosenau.

In the order of frequency of occurrence of these symptoms pain is the most common and is almost uniformly present; occult blood is the next important symptom. It is present in 83 per cent. of the cases. It is present in quite a large percentage of gastric and duodenal ulcers. Emesis is present in both gastric and duodenal ulcers, but for the most part is much more frequent in gastric ulcer variety than in duodenal ulcer. The fact that emesis is present in 54 per cent. of the duodenal ulcer cases is significant, as indicating how often the peritoneum is irritated by the depth of the duodenal ulcer, because of its close proximity to the serosa. In the stomach the muscularis mucosæ prevents the irritation of the peritoneum. So, you see, the reflex vomiting should occur rather in the case of the stomach ulcer. While the irritation should be greater in duodenal ulcer, the reflex vomiting should be greater in the stomach ulcer.

The location of the pain is also significant. That is the area of tenderness in 95 per cent. of the cases. Pain is present in 96 per cent. of duodenal ulcers as a distinct manifestation. It is present in 89 per cent. of those on the right side, that is, when the ulcer is to the left of the midline of the stomach, indicating where 85 per cent. of the ulcers occur. When it is to the left of that line the rule is that the patient will tell you just where the ulcer is in the stomach. When he locates the pain in a definite space on the left side, almost directly over the ulcer, you can feel quite

certain in making the incision over that spot. He cannot tell you definitely where it is when it is on the right side of the stomach. I am speaking of the left of a definite zone of the stomach.

Early in the course of this case, at the time of his original trouble, a diagnosis of appendicitis was made and a perforation of the duodenum was found. Appendicitis is the most common diagnosis of a perforation of a duodenal ulcer. They often and almost always diagnose an acute pain in the abdomen as appendicitis. It is difficult to make a differential diagnosis in that class of cases.

We never enjoy operating on a case a second time. Why? Because you expect to find adhesions, especially in this case. Why? Because he had a suppuration in his abdomen which had to be drained and redrained for a long time.

We will make the incision over the right rectus muscle as for a gall-bladder operation. We incise the skin, fascia, and anterior layer of the rectus aponeurosis. Then we displace the fibers of the rectus muscle outward and divide the peritoneum on the incline between two tissue forceps. We will expect to find adhesions. We are opening the abdomen with a little more caution than usual, because we are just fearful that we might come to a position where the intestine will be adherent to the anterior abdominal wall.

Now we are in the free peritoneal cavity. Note the dilated condition of the stomach. We are handling this with care, so that it will not rupture. On palpation I fail to find any evidence of gastric ulcer, but there is much evidence of a duodenal ulcer. The pyloric zone is embedded in a mass of adhesions, which accounts for the dilated stomach, and the duodenum is fixed to the under surface of the liver and to the gall-bladder, the result of the inflammation caused by the ulcer. The gall-bladder is edematous and reddened. Will I resect that? No. What is he suffering from? From an obstruction in the pyloric zone which is so significant as a symptom, as Dr. Mix brought out in his

discussion. The omentum is adherent down where the drainage-tubes were placed in the first operation.

I must ligate this omentum in order to free the transverse colon. I compress the omentum with a large clamp before the assistant ties the ligature into the crease made by that compression forceps. We never ligate that structure without first making a compression,—a firm compression,—and then we tie into the crease made by the angiotribe. That means what? That you force all the fat out of the bite of the ligature. You get a direct compression of the blood-vessels and you will not subsequently have a leakage and hemorrhage. That lets the transverse colon come up, so I can bring the under surface of the stomach into the field in order to make the approximation.

We will do a posterior gastro-enterostomy. First we locate the ligament of Treitz and that brings us to the first portion of the jejunum. I want to do the no-loop posterior operation. The air in the stomach has gone out into the jejunum, and the stomach is collapsed.

VISITING DOCTOR: Dr. Mix just made the statement that there was gas in the stomach.

DR. MIX: No. I said that there was no gas in the stomach. I said that persons with duodenal ulcer frequently complained of gas in the stomach, and that it was not gas in the stomach, but air pumped there in order to get rid of gas.

DR. MURPHY: There is a definite name for the pumping of that air in horses. They are called "cribbers." Exactly the same thing occurs in the human.

Here is the ligament of Treitz, and now I pick up the highest loop of the jejunum. Now I know I am right. Next I turn the omentum up over the epigastrium, so that the mesocolon can be held taut. The mesocolon is split longitudinally at a bloodless spot within the arch of the midcolic vessels, so that a portion of the posterior wall of the stomach can be drawn through. The lowest part of the greater curvature is selected for this purpose, so as to insure a good emptying of the stomach when the anastomosis is made. A few interrupted plain catgut sutures secure the stomach to the mesocolon, preventing its slipping back or

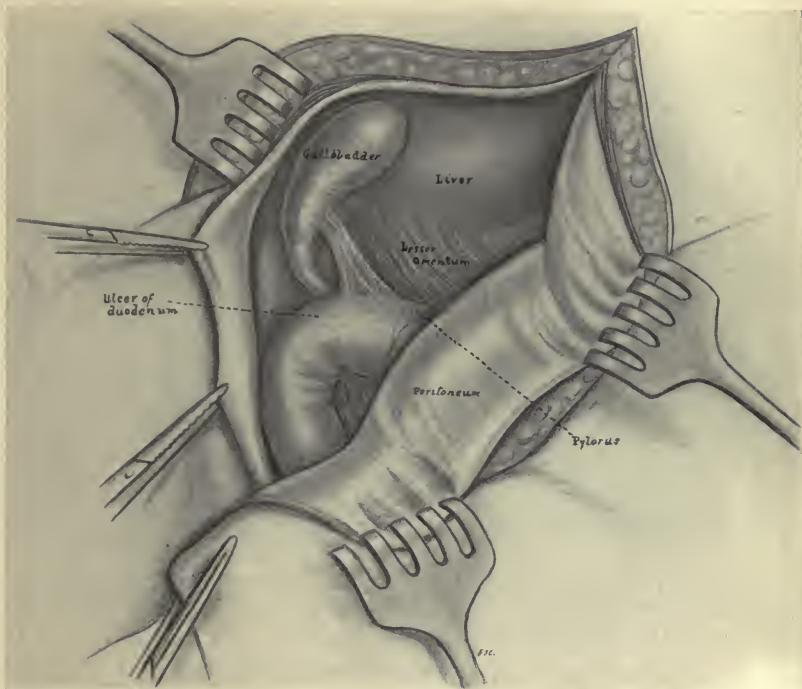


Fig. 105.—Healed ulcer of the duodenum with adhesions from duodenum to gall-bladder and under surface of liver.

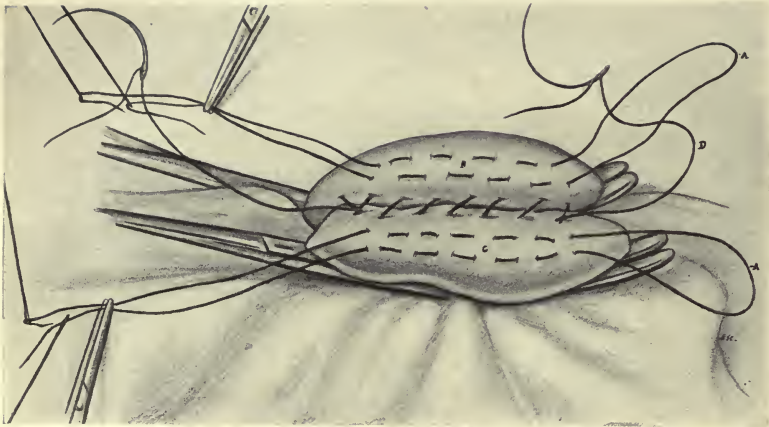


Fig. 106.—The parallel linen sutures (A) have been passed through the stomach (B) and the bowel (C) preparatory to incision of these organs for the insertion of the button. The posterior Lembert suture (D) shows well.

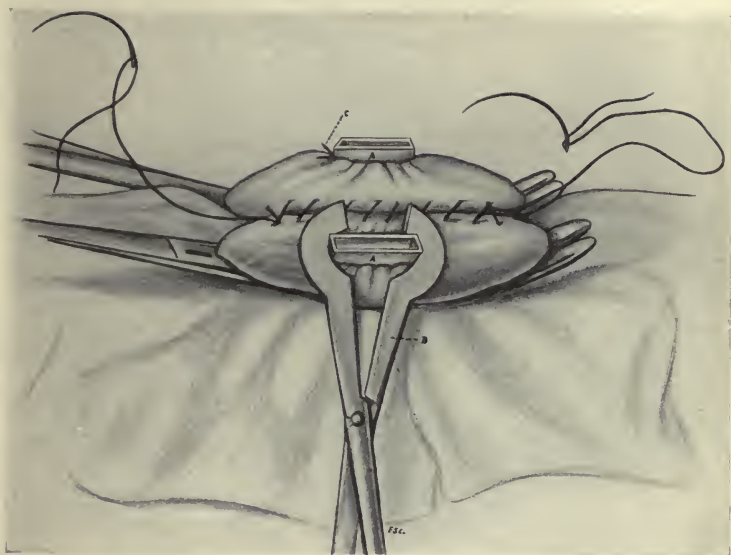


Fig. 107.—The halves of the button (A) are in place—the one in the bowel being held by a Hartmann clamp (B). Note that the purse-string suture on the upper half of the button is tied at the end of the cylinder (C).

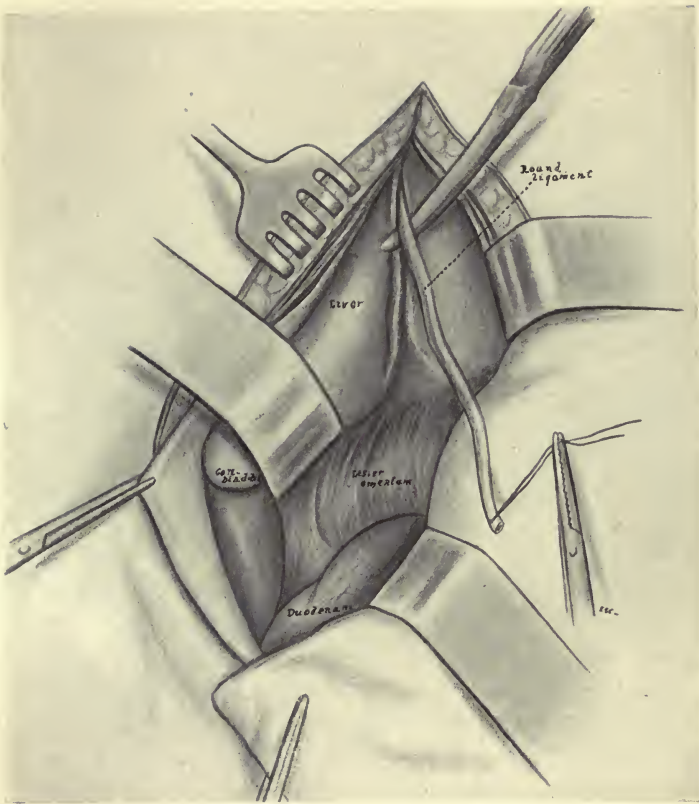


Fig. 108.—The adhesions to the gall-bladder have been freed. The round ligament of the liver freed from the falciform ligament preparatory to passing it around the pylorus. The location of ulcer in the duodenum shows plainly as a stellate scar.

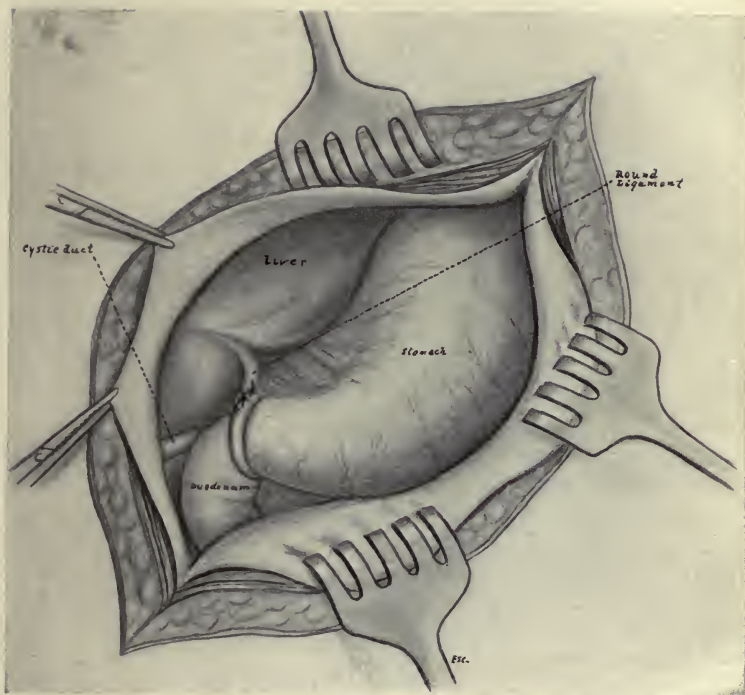


Fig. 109.—The round ligament has been passed around the pylorus from behind forward and the free end tacked to the proximal portion so as to occlude the pylorus.

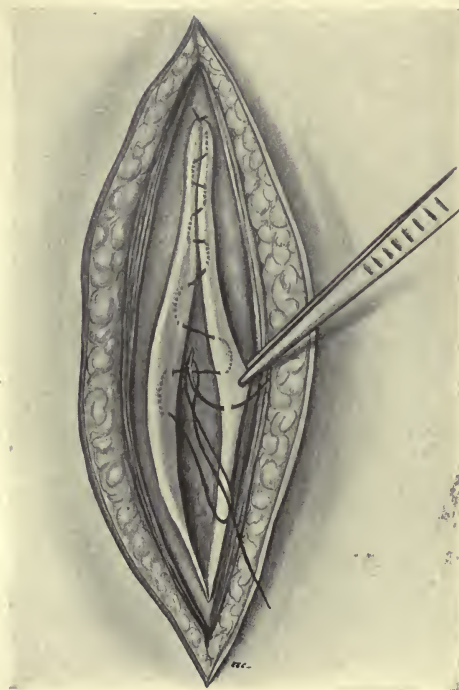


Fig. 110.—Showing Dr. Murphy's method of closure of the peritoneum by eversion of its cut edges, which prevents the occurrence of intra-abdominal adhesions.

becoming herniated. A curved gastro-enterostomy clamp is applied transversely to the protruding portion of the stomach.

The next step is to secure the jejunum. The highest loop is "milked" of its contents, and a second clamp is placed on a segment of the bowel. The two clamps are placed in a parallel position and are carefully wrapped about with gauze sponges to prevent soiling of the field of operation by stomach and bowel contents when these viscera are opened. The bowel and stomach segments are united posteriorly by a running catgut Lembert stitch. Then we take two glover's needles, $2\frac{1}{2}$ inches in length, threaded with both ends of a Pagenstecher linen suture, and pass them through all the coats of the bowel and stomach respectively $\frac{1}{4}$ inch apart for a distance of one inch and a quarter. Then an inch-long incision is made between them. The needles act as splints to support the viscus while it is being incised. Then the needles are drawn through simultaneously and held with a hemostat.

One half of an oblong Murphy button is placed into the bowel opening and the other half into the stomach opening and held with hemostats. The sutures are drawn taut around each half of the button respectively and tied, the knot being placed *at the end of the cylinder*—not on the side. That is essential to prevent subsequent leakage. The Hartmann clamp is placed on each of the cylinders to give full control of the button.

The gauze plugs which are always placed in the opening in each cylinder before operation are now removed, and the halves of the button are approximated and pressed together firmly. The original posterior Lembert stitch is now continued all around the line of union to relieve traction on the line of button approximation.

The clamps are removed, and the gastro-enterostomy is completed. The button operation is simple and safe. When performed properly, it is an absolute safeguard against subsequent leakage or hemorrhage.

As there is already a pyloric block here it will be unnecessary to operate for the closure of the pylorus. If we had to do this, we would have used the round ligament portion of the falciform

ligament of the liver for the production of a pyloric occlusion by passing it around the pylorus once or twice and suturing it accurately. A complete pyloric block can be established in a few minutes. This is, we believe, the safest means of occluding the pylorus.

The greatest danger that occurs in gastro-enterostomy is, first, pneumonia; second, hemorrhage into the lumen of the stomach or bowel. Pneumonia claims 16 deaths to the 12 from hemorrhage. Hemorrhage is entirely avoidable by the button. It makes a firm and uniform pressure on both the stomach and the intestine. We always use the oblong button in these cases. We went away from the button for a time, when we used the round button; then we did the suture operation; then we did half the cases with the suture and half with the button. Now we have come back to using the oblong button always unless there is some anatomic condition preventing its application, such as adhesions.

VISITING DOCTOR: Have you through personal observation of the button method and the exclusive suture method of doing gastrojejunostomy concluded that the button method is the best?

DR. MURPHY: Yes. I have concluded that from my own personal observation as well as from the experience of others. Sir Berkeley Moynihan uses the suture in all his operations. In Czerny's clinic at Heidelberg, Czerny being the father of the Czerny-Lembert stitch, the button has been used almost exclusively from the beginning. They have demonstrated that they have no hemorrhages and less regurgitation than those who use the suture method. The Mayos used the button for a great many years and then abandoned it for the stitch, which I did at one time, but I have come back to the button. That is a matter of election. One observation impressed me greatly in a recent trip abroad. I saw probably 200 men doing gastro-enterostomies in 25 or 30 different ways, and all were getting good results. Therefore it is not the instruments or methods used, but the cortication, that gives results. The only fatal hemorrhage I ever had was when I was using pyoktanin catgut.

The peritoneum is closed with a continuous catgut suture,

making an ectropion of the cut edges. The anterior aponeurosis of the rectus muscle is sutured with a double No. 2 plain catgut locked stitch. The skin edges are approximated with horsehair, but before doing so we put in four or five figure-of-8 silkworm-gut sutures to obliterate dead spaces. These sutures are tied over a gauze splint laid on the skin. That gives us a very firm and secure closure of the wound, with no possibility of skin necrosis from suture pressure or constriction.

In these cases we like to swab the wound with a 1 per cent. aqueous lysol solution after the rectus aponeurosis is sutured, because we have been in a possible colon bacillus field and the lysol stimulates the reaction of the tissues against these micro-organisms.

The usual plain sterile gauze dressing is applied, held in place with adhesive straps and an abdominal binder.

Let the record show that on examining the right hypochondrium the gall-bladder was found edematous and swollen; that the duodenum and pyloric zone of the stomach were adherent to the gall-bladder and to the liver, so that the stomach could not be elevated out of the abdomen; that a posterior gastro-enterostomy, no-loop operation, with the oblong button was performed in the usual way.

Let the record further show that Sister reports the sponges counted and correct.

Remember the original ulcer was not sutured. In an article on duodenal ulcer published recently the author showed that of 19 patients operated on for acute perforation of duodenal ulcer without suture, just drainage, 18 died immediately after the operation and the nineteenth died from a secondary abscess some weeks after. The proper treatment for a duodenal perforation is to make an incision; find the ulcer, suture it, and put in a drain down to it. It is not necessary to do a gastro-enterostomy, depending on how soon you see the patient after the accident. If you had the patient within a few hours, for instance, then you could do a gastro-enterostomy on the consideration that the life of the patient can be preserved. In the enormous number of duodenal perforations, if the operation is done early, suture the

ulcer, but the patients practically all die when the suturing is not done. I want to accentuate that, that it is necessary to close the point of leakage in order to prevent further leakage into the free peritoneal cavity.

VISITING DOCTOR: Dr. Murphy, do you operate on all cases of duodenal ulcer?

DR. MURPHY: All duodenal ulcers are operated if they are demonstrable. In the early stage, if you make the diagnosis, it is a surgical case at once, just like appendicitis, because even if the ulcer does heal it will recur, as in this case. Remember the average history of duodenal ulcer is twelve years of suffering; then they go to the surgeon for relief. That tells the story. That is not true, you understand, of gastric ulcer. I do not consider that all gastric ulcers are operative cases,—just the opposite,—and I feel that they should have medical treatment first, endeavoring to get them healed. It is only when they do not heal and remain healed that you have to resort to surgery.

We have three cases of this type in the hospital now in which we performed a gastro-enterostomy with the button. One patient passed the button on the fifteenth day, the second on the seventeenth day, and the third on the nineteenth day.

The oblong button is passed without colic or distress. That was not true of the large round button.

GOITER

A TALK ON THE EMBRYOLOGY, ANATOMY, AND PHYSIOLOGY OF THE THYROID

HISTORY

PATIENT, female, aged twenty-six, single, entered the hospital February 2, 1914, with the following history:

Family history negative to tuberculosis and carcinoma. One aunt had goiter.

Past history: Had chickenpox at four years of age, measles at eight.

Present complaint: About June 20, 1912, patient, while suffering from a severe cold, noticed some enlargement of the right side of her neck. She also had sharp shooting pains in the same side, which came and went at irregular periods; had difficulty in swallowing, was very hoarse, and breathing was labored when lying down. This condition continued for about two weeks, when all the symptoms subsided except the swelling and the hoarseness.

December 26, 1913, she first noticed that the right side of the neck, and also in the midline, was larger than before. The hoarseness increased, and her breathing became more labored when lying down. She has had no pain in the neck of late, no chills, no tremor, nor have her eyes changed in any way.

Menstruation began at fourteen, regular, every twenty-eight days, three days' duration, one or two napkins a day, no pain. Last period ended January 27, 1914.

COMMENTS AND OPERATION

DR. MURPHY (Operation, February 16, 1914): Before we take up the discussion of this particular case, let me remind you briefly of the embryology of the thyroid, its histology as well as

its anatomy, because with a proper understanding of these the pathology of the thyroid is simplified very much. The thyroid is derived from the branchial epithelial bodies, structures which arise either as thickenings or outpouchings of the epithelium lining the lower portions of the inner branchial furrows. Five lateral pairs of these structures and one single unpaired median body are developed. The latter gives rise to the greatest part of the thyroid. It is situated immediately behind the anterior portion of the tongue, at the apex of the groove between this and the posterior portion. It appears first as a slight depression, and as this depression deepens its extremity becomes bilobed, and later separates from the floor of the pharynx. The original point of origin remains permanently as a circular depression on the tongue—the foramen cæcum.

Still later the bilobed body passes down into the neck and assumes the shape of a solid, transversely elongated mass; into this substance trabeculæ of connective tissue extend, dividing it into a network of anastomosing cords which later divide transversely to form follicles. Some embryologists claim that a cylindric outgrowth arises from the anterior surface of the mass, and, extending up into the neck a varying distance, forms the so-called pyramid of the thyroid or the thyroglossal duct. It does not usually persist until adult life, although His claims that it is the remains of the stalk connecting the thyroid with the floor of the pharynx.

Two other pairs of bodies are also formed—the parathyroid bodies. One of these pairs arises as a thickening of the dorsal portion of the fourth branchial groove, and the other comes from the corresponding portion of the third groove. These bodies later become separated and come to lie on the dorsal surface of the lateral portions or lobes of the thyroid, usually one at each pole of the gland, although their final resting-place is by no means a definite one or a uniform one in all cases. The cells of these bodies do not become divided into cords by the ingrowth of connective tissue, nor do they become separated into follicles, so that they are readily distinguishable by their structure from the thyroid. They are of a yellowish or rather buff color.

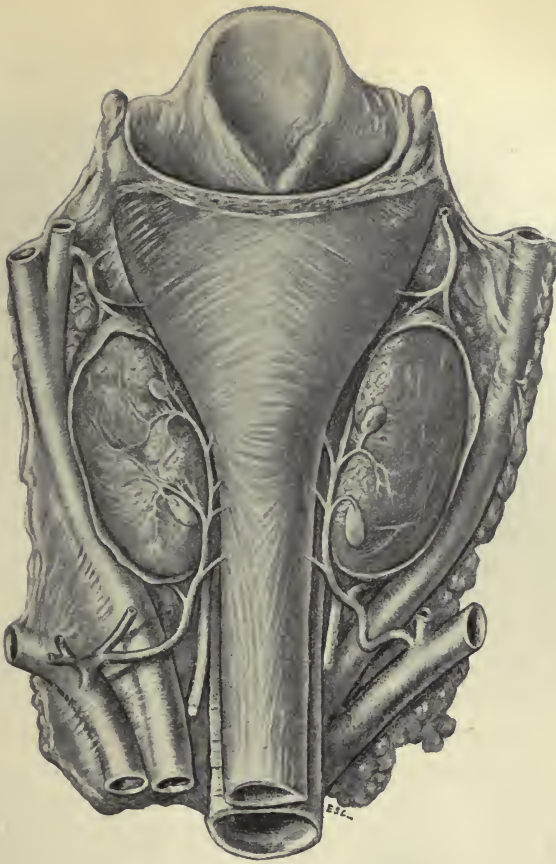


Fig. 111.—The position of the parathyroids in the human, and the relationship of the thyroid arteries and the recurrent laryngeal nerve (after Halsted and Evans).

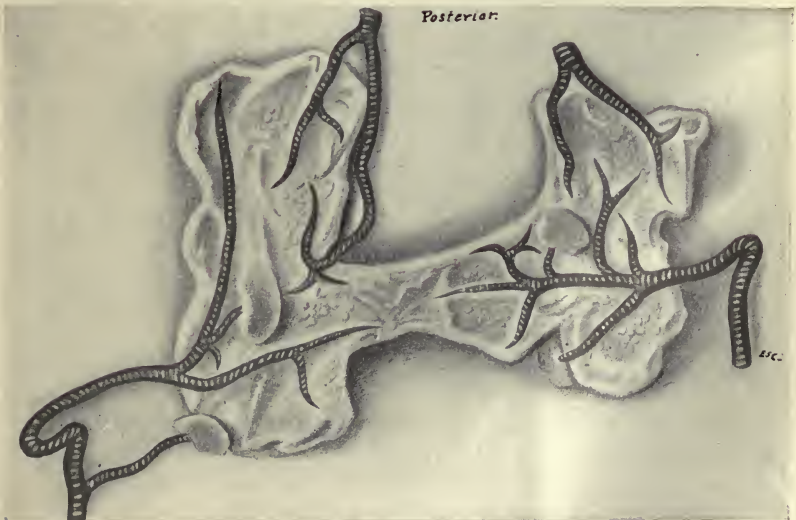


Fig. 112.—Arterial supply of the parathyroids. Posterior view (after Geis).

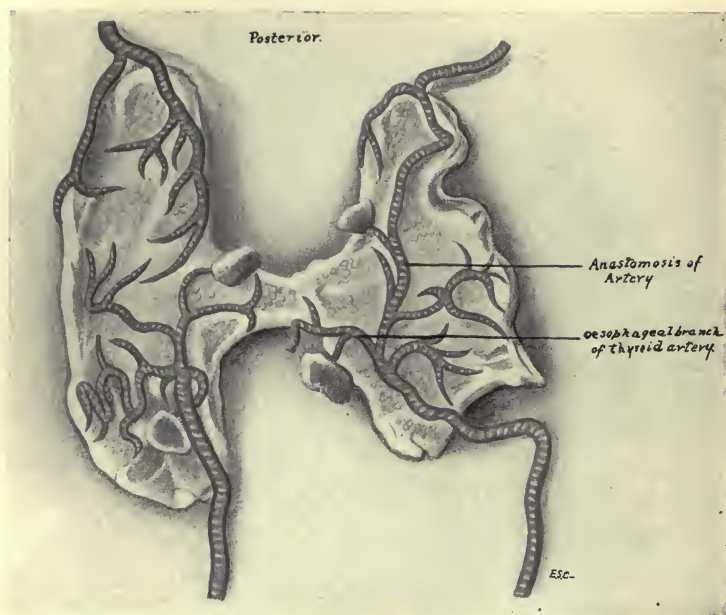


Fig. 113.—Arterial supply of the parathyroids. Posterior view (after Geis).

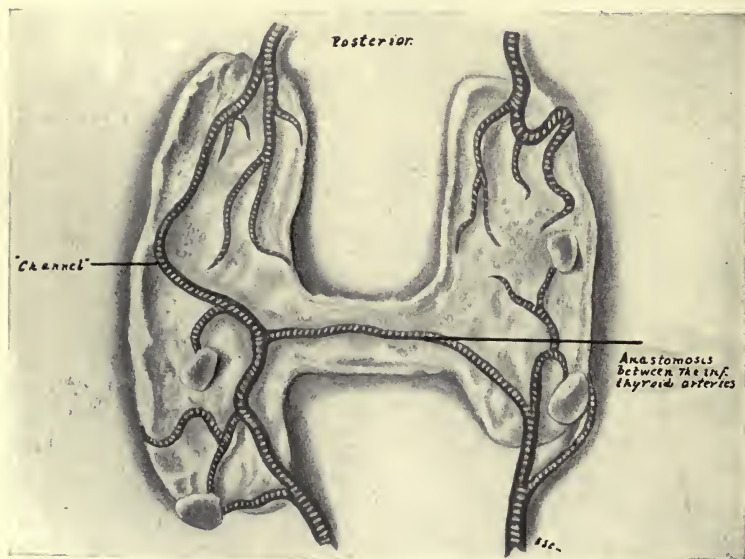


Fig. 114.—Arterial supply of the parathyroids. Posterior view (after Geis).

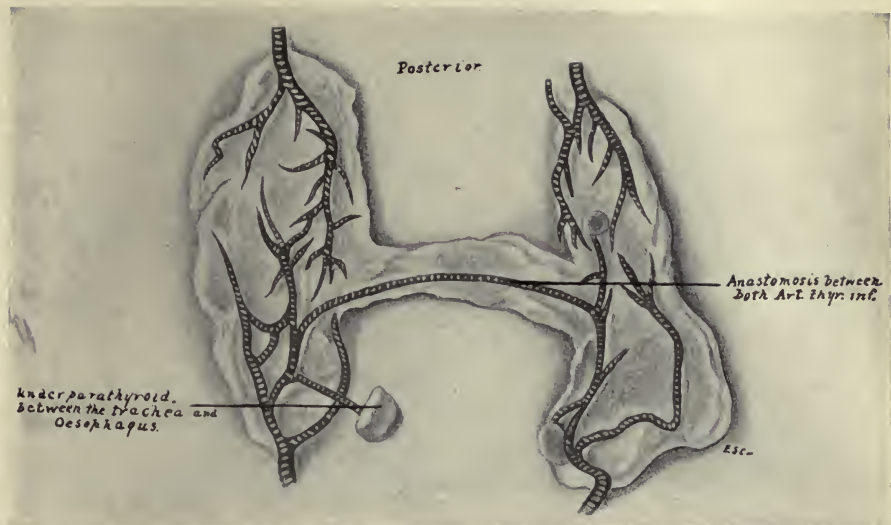


Fig. 115.—Arterial supply of the parathyroids. Posterior view (after Geis).

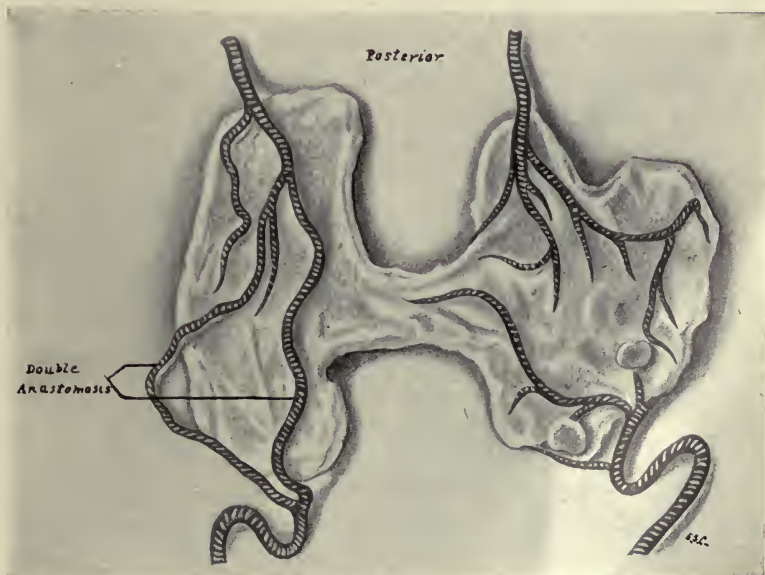


Fig. 116.—Arterial supply of the parathyroids. Posterior view (after Geis).

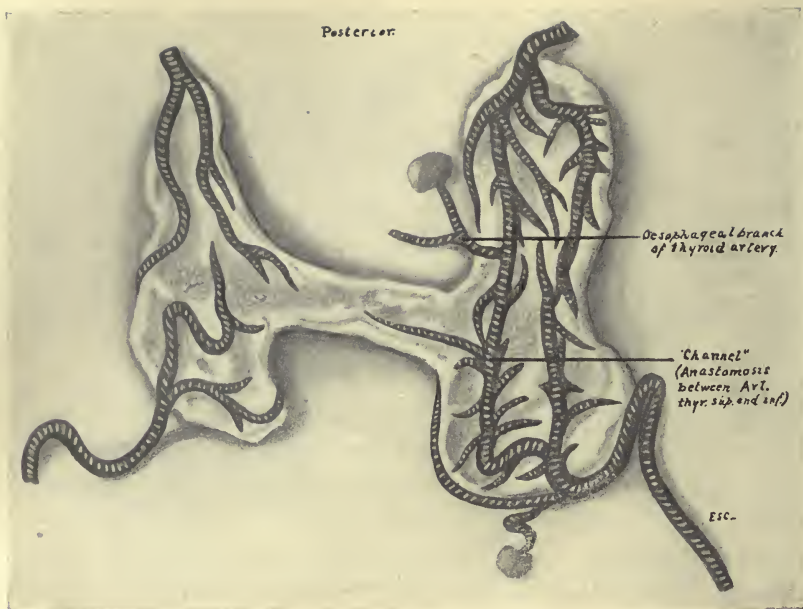


Fig. 117.—Arterial supply of the parathyroids. Posterior view (after Geis).

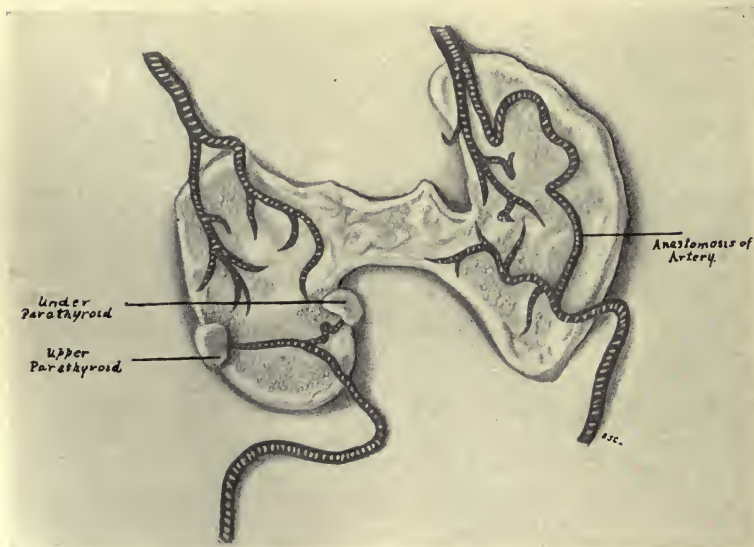


Fig. 118.—Arterial supply of the parathyroids. Posterior view (after Geis).

In the adult the thyroid consists of two lateral lobes and a median lobe—the isthmus. It is situated at the front and sides of the neck, and extends upward on each side of the larynx. Although the thyroglossal duct is usually obliterated before birth, becoming a cord of epithelium, the lower portion not infrequently remains open for a short distance. The gland is inclosed in a closely adherent, thin, connective-tissue capsule. Ordinarily the lower portion of the gland, when the head is extended, is about one inch above the upper margin of the sternum; when the head is flexed, it is at the level of the upper border of the sternum, or even below or behind it. At the inner and posterior part of each lateral lobe is the hilum, the point at which the inferior thyroid artery passes into the gland, and here also the recurrent laryngeal nerve comes into close contact with the gland lying in the space between it and the trachea and the esophagus.

The thyroid is covered by the deep fascia and skin, the sternomastoid, anterior belly of the omohyoid, the sternohyoid, and sternothyroid muscles. These anatomic relations can be readily seen in the accompanying cuts of the thyroid and its environment.

The gland is supplied by the superior thyroid arteries (branches of the external carotids), and the inferior thyroid arteries (branches of the thyroid axis of the first part of the subclavian). Occasionally there is a third supply from a median thyroid artery, which, when it exists, is given off from the innominate artery, from the arch of the aorta, or from the common carotids. The superior thyroid arteries enter the gland at the upper and outer poles respectively. The relation of the inferior thyroid artery to the recurrent laryngeal nerve is of importance. Usually the main trunk of the artery passes behind the nerve; sometimes it breaks up before reaching the nerve, in which case one or more of its branches may pass in front of the nerve. The larger branches of the thyroid arteries are beneath the capsule and on the surface of the gland; the smaller branches pass into the interior of the gland.

The veins accompany the arteries. The lymphatics are large and numerous.

The parathyroids lie usually on the trachea or on the pos-

terior surface of the lateral lobes of the thyroid, but are always completely separated from it by connective tissue. They usually number four, two above and two below. They are not much larger than an orange-seed, but are easily recognized by their color.

Histologically, the thyroid consists of blood and lymph-vessels, epithelial cells, and connective tissue. Remember that it is one of the ductless glands. The capsule sends trabeculæ down into the gland, dividing it into follicles or acini, which are lined by a single layer of cubical cells. These follicles are closed cavities. They are of varying size and shape, and contain a characteristic fluid known as colloid, which is secreted by the epithelial cells. Bearing in mind the structure of the thyroid, one can easily outline the conditions to which the gland may be subject from a pathologic point of view. Before entering on that phase of the question I want to refer to the accessory thyroids, the small isolated masses of thyroid tissue found inside or outside of the normal capsule of the gland, anywhere in the cervical fascia, in the root of the tongue, in the mucous membrane of the larynx, or the trachea or bronchi. It is believed that these accessory thyroids may, as a compensatory condition, assume all or a part of the function of the thyroid itself, and that is undoubtedly what occurs in those cases where the thyroid has been removed in its entirety and the thyroid metabolic function continues unchanged.

Another point which I want to emphasize is that the blood-vessels of the thyroid are of unusually large size considering the size of the gland supplied, and there are many anomalies of distribution, so that in operating on the thyroid for any condition we must be exceedingly careful and look for every vessel to make sure that it has been found, clamped, and ligated. The venous circulation is more irregular in its distribution than the arterial, and the hemorrhage from the veins often far exceeds that from the arteries.

We have had placed on the board this morning a synopsis of the pathologic conditions encountered in the thyroid.

THE THYROID GLAND

Congenital Anomalies:

- Complete or unilateral defect.
- Abnormal lobulation.
- Accessory thyroids.
- Premature atrophy.
- Hypoplasia.
- Congenital enlargement.

Circulatory Disturbances:

- Hyperemia.
 - Arterial.
 - Puberty.
 - Menstruation.
 - Pregnancy.
 - Venous (congestion).
 - Exophthalmic goiter.
 - Struma vasculosa (angioma).

Inflammations, Infections, and Parasites:

- Acute thyroiditis.
- Chronic strumitis.
- Acute exudative—
 - Trauma.
 - Infection.
- Chronic productive (struma fibrosa).
- Tuberculosis.
- Syphilis.
- Cysts—echinococcus.
- Actinomycosis.

Retrogressive Changes:

- Atrophy.
- Degenerations.
 - Fatty.
 - Coagulation necrosis.
 - Hyaline.
 - Calcification.
 - Colloid.
 - Amyloid.

Progressive Changes of the Secreting and Vascular Elements:

- Hyperplastic.
- Parenchymatous.
- Cystic.
- Hemorrhagic.
- Vascular.

- Calcifying.
- Colloid or gelatinous.
- Exophthalmic.
- Tumors:
 - Benign.
 - Adenoma.
 - Cystic.
 - Interacinous cyst.
 - Papillary cystadenoma.
 - Fetal.
 - Cylindric cell.
 - Fibroma.
 - Osteoid chondroma.
 - Chondroma.
 - Osteoma.
 - Teratoma.
 - Malignant.
 - Sarcoma.
 - Round-cell (small and large).
 - Spindle-cell.
 - Giant-cell.
 - Alveolar.
 - Angiomatous.
 - Mixed type (fibro-, chondro-, osteo-).
 - Perithelioma.
 - Endothelioma.
 - Carcinoma.
 - Carcinoma simplex.
 - Carcinoma medullare.
 - Carcinoma scirrhusum.
 - Carcinoma myxomatodes.
 - Cylindric cell.
 - Squamous cell (epithelioma).
 - Papillary cystic.
 - Mixed sarcoma and carcinoma.
 - Mixed carcinoma and perithelial angiosarcoma.
- Cysts:
 - Hemorrhagic.
 - Grumous cysts.

First on the list are the congenital anomalies. Complete or unilateral defect is rare. The isthmus is more often absent than are both lobes or either lateral lobe. Abnormal lobulation is ob-

served more frequently. The accessory thyroids are common, and may be found at a considerable distance from the gland itself, and have been known to give rise to tumors. Premature atrophy or even hypoplasia is the condition responsible for cretinism. Congenital enlargement may lead to death from pressure on the air-passages. The single gland in the base of the tongue may be the only thyroid tissue in the neck, and that may undergo the various pathologic changes observed in the thyroid when in its normal anatomic position. The removal of this single tumor may lead to grave complications.

Cretinism is a very uncommon condition. Still sometimes one sees a whole family of cretins. I recall such a family, something like five members, in my early service at the Presbyterian Hospital. They were put on thyroid treatment, but did not remain under observation, so that I could not determine whether or not they were cured or improved. You know in animals you can stop growth in height by taking out the thyroid. When you remove the thyroid from a litter of greyhound pups they develop into "dachshunds," depending on how much thyroid you administer as food to preserve life after the gland is removed.

When you come to consider the pathology of the thyroid, all you have to bear in mind is the three elemental tissues that enter into the anatomic formation of the gland—the connective tissue, that holds the gland together, the epithelial or specific enzyme cells, and the blood and lymph vessels. These three histologic elements are involved separately or combined to produce the enlargements. Let us consider the lesions of the connective-tissue group. Enlargement of the gland may be the result not alone of connective-tissue hyperplasia, but of connective-tissue infection, usually of the hematogenous or metastatic type. Besides the acute enlargement and sensitiveness of the gland, there is often a high temperature, sometimes delirium, and a low "typhoid condition." It may come as a sequence of tonsillitis, of measles, of scarlet fever, or any other infectious disease. You have an infection, a round-cell infiltration, and a hyperplasia of the connective tissue (acute thyroiditis). The patient is intensely sick, and may die in that stage of the disease. If the

patient survives, the connective tissue matures and there is then a fibrosis with subsequent contraction just the same as we have in cirrhosis of the liver or in any organ where we have an excessive quantity of connective tissue—first, a hyperplasia, and the secondary stage is always that of a contraction. That contraction may go on to the stage of complete absorption and destruction of the specific gland cells, to the production of a hypothyroidism, appearing as a myxedema. The patient commences to show a thickening of the skin on the forearm, the arm, and the hands; the face commences to change its expression; the lips become a little thicker; the skin of the face becomes thicker, but not the type of thickness we see commonly with nephritis. It goes on and develops a myxedema, and still the gland may be no larger in appearance than a normal one; it may be even smaller or harder. As the disease advances, the patient develops an anasarca, the axillary and suprapubic hairs disappear, the eyebrows become smaller, and the hair on the head very materially less. The patient has recurrent attacks of diarrhea, and occasionally chills and fever. The intellect becomes slower in response, etc. (This condition is rapidly relieved by the administration of iodothyryn.) The other lesions of the connective tissue are the tumors, benign (see synopsis) and malignant in type—sarcoma. That is all there is to the connective-tissue lesions.

Let us take the next structure that is subject to disturbances—the vascular structure. What are the lesions that occur in the vascular structures? There may be a hyperplasia, an excessive development of the arteries throughout the gland causing a tense, pulsating tumor, not only pulsating up and down, but a lateral expansion pulsation. That is the most dangerous pathologic condition you can possibly encounter in removing the gland. That is why it is essential one should know these lesions.

A dilatation of the veins in the gland may cause a greatly swollen neck. When the patient coughs, the neck will enlarge 20 to 50 per cent. above the average size, just as though you had inflated it with an air-pump. When you put your hand on it and compress it slowly, it will reduce in size. If you ask the patient

to inhale, you get a minus pressure on the chest, and it will greatly shrink. After a deep inhalation ask the patient to cough and the veins fill up again. It is a splendid point in making the differential diagnosis between the arterial vascular and the venous variety of goiter. In the venous variety you will find difficulty on account of hemorrhage in removing the capsule. In place of being able to shell out the capsule, going along a square inch without encountering any vessels, you will have vessels passing from the capsule every $\frac{1}{4}$ to $\frac{1}{8}$ inch. It is just like a sieve.

The next vascular variety is a combination of the two, the arteriovenous type, where the veins are distended and the arteries are pulsating. That is a still more difficult type to handle. When you run your finger around on the side of the gland to elevate it, you tear off a small number of vessels, which results in severe bleeding. Put on a compress, and slowly raise it up as you put on the ligature.

The lymphangioma is a dilatation of the lymphatics, and causes the lymphatic type of goiter. There is a hyperplasia of the lymphatics throughout the gland, and the condition may extend into the neighboring tissues, making it difficult to remove the gland. That completes the vascular variety, the arterial, venous, and arteriovenous variety, and the lymphangioma.

Now, then, what additional anatomic structure remains in the gland to deal with? Just one: the glandular or secreting element of the thyroid. What are the lesions there? A general hyperplasia, a parenchymatous goiter. The gland follicles or acini are enlarged. The cells lining the acini are increased in number; the number of acini is increased, and the number of cells in the interstitial tissue is increased, without any manifestation whatever of a hyperproduction of the gland secretion. There is no manifestation of a hyperthyroidism. Whether there is more material produced or not we do not know, but if it is produced, it is neutralized in the metabolism, because the patient has no evidence of a thyroid intoxication. That is the average enlarged gland you see at puberty, during gestation, and after parturition.

The next variety in the order of frequency of occurrence is the colloid goiter. How does a colloid goiter differ from a normal gland? There is a loss of balance between secretion and absorption. When the process is advanced, you have the typical colloid goiter, a number of miniature excessive retention accumulations. This mass of colloid cysts means just a production beyond the power of absorption. When it is circumscribed, it is an encapsulated colloid goiter. After the tubules are distended with colloid material the pressure on the columnar cells causes them to flatten; this change of conformation in the cells alters the character of the secretion to a serous material. As this accumulates a cystic goiter develops. The process may extend all through the gland, and multiple cysts are formed, or if it is circumscribed, a large monolocular cyst may develop.

Now as to the tumors. A circumscribed or encapsulated hyperplasia or increase in the glandular elements is an adenoma of the thyroid, and, as shown by our table, there are varieties of adenomas, as to point of origin, size, and clinical course. There are other benign tumors of the glandular element, and one malignant variety of carcinoma, none of which we will discuss at this time. However, they are included in the synopsis on the board.

Of particular interest is Graves' disease. It is a manifestation of an excess of thyroid secretion, not necessarily above normal, but above that which is utilized or neutralized in the metabolism. That may be associated with what? A change in the gland. It may be associated with a hyperplasia of the epithelial cells, a hyperplasia limited to a circumscribed zone, or a perceptible enlargement of all of the gland. You may have a patient who has had an ordinary colloid goiter or an ordinary adenomatous goiter, and that patient may have gone along for years and then suddenly manifest evidences of Graves' disease. That is due to the fact that there is a separate area which has taken on an acute secretion and is producing material more rapidly than it is utilized. It is merely grafted onto an ancient goiter. When you extirpate glands of this type you will find the old lesion clean cut, and you will find a new lesion, an acute vascularization, often in

an area not larger than a walnut, with the most severe symptoms of Graves' disease. Commonly it develops as an advanced condition if it is an acute enlargement. The vascular supply is increased, so that on physical examination you may hear a bruit over the gland. The pressure of the blood is also increased. The "why" of that has not been explained yet, but the fact remains that there is an excess of blood and that the gland pulsates. What effect does hyperthyroidism have on the constitution? Tremor is an early symptom. Often, however, long before the tremor is present, the patient will tell you, "I feel as though I am shaking all over inside." He has a feeling of tremor in every portion of the body. Then there are the tachycardia, loss of body weight, exophthalmos, an enlargement of the thyroid, and finally disturbances of the digestive tract.

That outlines practically the whole picture of the thyroid gland pathology. But we have said nothing at all of the etiology, nothing as to why the gland is enlarged. We have considered the structures which change in enlargement of the gland. What is the etiology of an enlargement of the gland? The etiologic factors of acute infection are suppuration in some other position, which metastasizes to the thyroid; the most common is streptococcic epidemic pharyngitis and tonsillitis. Of the chronic types, the drinking-water probably plays an important rôle. There are several theories; one is that there is some substance in the drinking-water, or in excess in the drinking-water, which causes goiter, and formerly it was thought the water lacked some element, which deficiency caused the hyperplasia. In Switzerland, in particular, they have in certain localities a drinking-water that they claim causes the goiter. In some districts the entire population have goiters. In the water shipped from these districts to the laboratories in Berne and Lausanne they found when they gave this water to young rats and guinea-pigs that it produced goiter. They also found that the river from which this water was taken passed through a little town, that on one side of it there was practically no goiter and on the other side it was in every household. Furthermore, they found by taking the water from the side where goiter was not present

through a tunnel over to the other side, that there were no new goiters originating. They found when the water was boiled it failed to cause goiter in the experimental tests. That proves that there is something in connection with the water-supply in this part of the country that stimulates the production of goiter. There is apparently in the acute Graves' cases an additional nerve element, the element of shock or over nerve tension. The patient learns of some serious situation and is greatly frightened or becomes embarrassed in some way; suddenly there appears an enlargement of the thyroid; this phase is too common to be a mere coincidence. An illustration of that type was the case of a young lady who learned of some conduct on the part of her father that was scarcely believable. She suddenly developed an enlargement of the gland; her pulse-rate was 140 to 180; the eyes protruded; great tremor and all the manifestations of Graves' disease came in about four weeks. She was in so serious a condition that I hurried her out of my office, fearing she might die. The case was inoperable on account of its severity. We sent her home, put her on rest and arsenic treatment, and in fourteen months the goiter had entirely disappeared, but she kept her bed for fourteen months at absolute rest, with an ice-bag on her neck and one on her heart. The gland has diminished to normal size, but is very hard, due to the contraction of the connective tissue. She has been well for four years.

The case we are going to operate on today is one of adenoma involving the right lobe. The patient has been in the house for about a week, being prepared for operation. We always like to have them rest in bed, remaining absolutely quiet for that length of time so that they will be in the best possible condition for operation.

We will expose the gland through the usual transverse curved Kocher collar incision, which affords free access and leaves an almost imperceptible scar. The skin, platysma, and deep fascia are divided and dissected and reflected well up. The thyro- and hyosternal groups are there divided and reflected downward; all vessels are secured quickly, if possible before they are cut. One of the prime essentials in any operation on the thyroid is to have

a sufficient number of hemostats, and to get them on quickly to prevent the loss of more blood than is absolutely unavoidable.

Retracting the wound edges, the gland comes into view, but in order to secure a good exposure we must divide the sternohyoid and sternothyroid muscle groups a little more to the side. Hemostats are placed on each group separately, as high up as possible, in order to preserve their nerve supply, and then the muscles are divided between two hemostats. We must also make a partial division of the right sternomastoid; that gives us a splendid exposure.

There is the right carotid artery on the outside, right in the field. I will commence and free this gland from behind and elevate it, so as to lift that lobe up. It is elongated, going backward well down on the esophagus. Now I will roll this gland over toward the left side and wipe off the capsule, which I have divided over the center of the gland. I now expose the upper pole, grasp the superior thyroid vessels, and divide them between the forceps, and the pole is free. I commence to elevate it from below, so as to come around in the direction of the lower lobe. Here is where one encounters the recurrent laryngeal nerve, right on the gland. We have not yet seen a parathyroid, but we are always looking for one. Now we shall go under the right lobe, checking hemorrhage as we go along, making sure to secure all vessels before they are torn or divided. Special care is taken in dealing with the inferior thyroid; because of its size and relation to the nerve it should be secured in the capsule $\frac{1}{2}$ to $\frac{3}{4}$ inch from the gland where it is free from the nerve. We prefer to cut through the substance of the gland, leaving the part that is attached to the trachea, because by doing so we insure the safety of the underlying structures, especially the recurrent laryngeal nerve.

We have now completed the removal of this lobe and a portion of the isthmus by clamping the isthmus to the left of the midline with this small curved Mayo clamp which I prefer to the Kocher, then divide with scalpel and cauterize the surface and ligate the isthmus. The next step is to ligate all bleeding points. We have on here a large number of hemostats, and we

are going to ligate every place where there is a hemostat, because if we do not do so, no matter what we think about it, it is going to bleed; that is the assumption. By using the actual cautery on the cut surfaces of the isthmus you prevent leakage of thyroid material from that gland. I always sear that surface with the actual cautery, not because I depend on it as a hemostatic, but to prevent the seepage of thyroid secretion into the wound.

Now we are ready to reunite the divided muscles in their normal anatomic relation, using for that purpose a fine catgut. You will note what a splendid reposition of structures that makes. There is scarcely any deformity of the neck; the deep fascia was also reunited with catgut and the skin wound is closed with horsehair. A rubber tissue drain extends clear to the deepest point of the cavity and comes out in the midline; this drain will be removed in twelve to thirty hours as soon as the wound secretion has ceased.

The wound is dusted with bismuth subiodid and a large dressing is applied. The patient will be put in bed in the semi-sitting position. The horsehair stitches will be removed in ten or twelve days.

Let the record show that this goiter had in it the following conditions: First, a circumscribed adenoma; second, a circumscribed adenoma undergoing cystic degeneration or breaking down into a cyst; third, a general hyperplasia all in the right lobe.

You see in reconstructing that neck we just reverse the order in which we open it, uniting first the deep layer of muscle and then uniting the margin of the sternocleidomastoid on the right; after that we unite the platysma and then the skin. We occasionally use clips in the skin. There is no deformity left in the neck when one does it in that way. The derma should always be steadied when the incision is made so as not to bevel the skin. When the skin is not cut *perpendicular* to the surface there is greater tendency to fibrous deposit in the scar and more frequently a keloid thickening. Goiter is now one of the safest of all operations except in the Graves type, and this can be made safe by proper selection of the time for operation.

We always use nitrous oxid ether oxygen anesthesia. Morphin and atropin are given one-half hour before operating.

TUBERCULOSIS OF KIDNEY: NEPHRECTOMY

HISTORY

CASE I.—Patient, Mr. S., male, aged twenty, enters the hospital because of occasional cramp-like pains, beginning sometimes in the region of the umbilicus and radiating to the left side, and at other times beginning in the region of the left kidney posteriorly and radiating anteriorly to the midline. He also has night-sweats and frequency of urination, having to get up four or five times at night to urinate. He has some trouble in starting the flow, sometimes ten minutes intervening before the flow starts. He also has slight pain and a burning sensation at the meatus urinarius at the beginning of the flow, but no pain in the bladder region. Previous to the onset of this trouble he was perfectly well.

Present trouble began about two and a quarter years ago, when the patient began to have occasional dull pains in the region of the left kidney. This pain persisted for a couple of days and then ceased, only to recur after a short interval. He never had a chill. The urine did not contain either blood or pus. No nausea or vomiting; no increase in frequency of or pain on urination; no pain in the bladder. He consulted a doctor, who diagnosed his ailment as a nervous trouble for which he gave him treatment for three months, but the pain still persisted, and, in fact, was somewhat more severe.

Three months later he consulted another doctor, who made a skiagram of the left kidney and diagnosed the case as one of ulcer of the stomach. He treated the patient with medicines for nine or ten months, but gave him no relief. Two months after this he consulted a third doctor, who also diagnosed the trouble as ulcer of the stomach, and put the patient on "stomach treatment" for sixty days. At this time he first noticed that the

urine was cloudy. He was sent to this hospital eight months ago (February, 1913) with a diagnosis of ulcer of the stomach, and operation was advised by his doctor.

He was examined by Dr. Murphy, who found a slight bulging in the left kidney region posteriorly, a little to the left of the mid-line, and on deep fist percussion elicited a marked hypersensitivity over the left kidney. A tuberculin test was made, and it was superlatively positive. The radiograph was negative. At this time the patient was having night-sweats, and following the tuberculin test he passed blood in the urine for three or four days, after which no more blood appeared. He does not know whether he had any fever preceding the test, but he noticed that he was urinating more frequently and that he had to get up three or four times at night to urinate, although he was passing only small quantities of urine each time. He was also having some trouble starting the flow, with pain and burning at the neck of the bladder.

He has been under tuberculin, cod-liver oil, and fresh-air treatment since this time. He never had any cough or bloody sputum. Two or three times in the course of the disease the pain radiated down into the scrotum.

Previous history negative. Denies venereal infection. No lues.

Family history: Has one brother and three sisters, all living and well. Parents living and well. Had one uncle who died of pulmonary tuberculosis.

COMMENTS AND OPERATION

DR. MURPHY (October 8, 1913): This patient stated that the first symptom was pain. That is probably true so far as he was able to determine, but pain is rarely ever the primary symptom of a kidney tuberculosis. The primary manifestation of all the tuberculous lesions is usually the constitutional condition of the patient preceding the local manifestation of pain. That is so uniform and so common an occurrence and it is yet usually erroneously diagnosed as biliousness, malaria, etc., for months and months before the local lesion in the kidney is recognized, before the local lesion in the bladder is recognized, before the local lesion

in the epididymis is recognized, before the local lesion in the peritoneum, or before the local lesion in the lung, is recognized. It is so uniformly present that whenever you get a history of a gradual general deterioration of the patient, the first question you should ask yourself is, is this the initial stage of a tuberculosis?

Tuberculosis of the kidney is usually a hematogenous infection, the primary focus being located elsewhere in the body, especially in the bronchial or mesenteric lymph-nodes, and not necessarily active in its clinical manifestations. In some cases, however, the infection is an ascending one, by way of the ureter, from the bladder, prostate, seminal vesicles, epididymis, or testis. In rare instances it is due to extension from the adrenals or other paranephritic tissues.

The condition manifests itself clinically either as an acute miliary tuberculosis, often a part of a generalized systemic process, or as a chronic local tuberculosis. If the case is one of hematogenous infection, the tubercle bacilli reach the kidney through the renal artery, and they may lodge in any part of the vascular system of the kidney; most often, however, in the glomerulus, and clinically it has been found that the cortex of the kidney is in most cases involved first, next the pyramids, which is significant of a lodgment of the bacilli in the tubules, and, lastly, the pelvis.

I have placed on the board a synopsis to aid you in following my discussion of this subject this morning:

SYNOPSIS OF TUBERCULOSIS OF KIDNEY

Occurs in 5 to 10 per cent. of all early cases of tuberculosis. Practically in all advanced cases.

A. *Route of invasion:*

1. Hematogenous: From lungs, bones, intestinal tract, lymph-nodes.
2. Urogenic: Ascending from other parts of genito-urinary tract.
3. Extension from neighboring structures (adrenals, spinal caries, tuberculous empyema, intestines, peritoneum).

B. Types:

1. Acute—Usually secondary (generalized systemic tuberculosis). Primary focus may be small—in one or both kidneys; usually a miliary tuberculosis beginning in the cortex.
2. Chronic—Hematogenous or ascending; one (80 to 95 per cent. early cases; 37.6 per cent. late cases) or both (62.3 per cent.) kidneys involved; right kidney more often than left.

C. Forms:

1. Massive, caseous or ulcerative (abscesses), cortical.
2. Pyramidal ulcerative.
3. Miliary (nodular) cortex and pyramids.
4. Pyelitic ulcerative.

D. Accessory etiologic factors:

Inherited tendency to tuberculosis.
Chronic pyelitis.
Cystitis.
Urethritis.
Gonorrhea.
Congenital deformities.
Pregnancy.
Floating kidney.
Calculus.
Hypernephroma.
Hydronephrosis.
Trauma.

E. Symptoms:

1. Acute miliary renal tuberculosis: characteristic symptoms referable to kidney rarely present, with the possible exception of recurrent hematuria.
2. Chronic or primary renal tuberculosis:
 - A. *Urine:* Increased frequency and painful urination.
Incontinence; diminished in amount; normal or polyuria, especially nocturnal; oliguria or even anuria; acid reaction; albumin; hematuria; pyuria; tubercle bacilli in urine; casts.
 - B. *Local swelling and pain.* Great sensitiveness to fist percussion (the Murphy test).
 - C. *Constitutional disturbances:*
Fatigue, malaise, dyspnea, anemia, emacia-

tion, cachexia, anorexia, digestive disturbances, chills, fever, profuse sweats.

F. *Differential Diagnosis:*

Calculus; malignant tumor; pyelitis; pyelonephritis; hypernephroma.

G. *Treatment:*

Prophylactic	{ Medicinal (tuberculin). Climatic. Hygienic.
Surgical	{ Nephrectomy. Nephrotomy. Nephrostomy. Partial resection.

H. *Operative mortality:* 3-10 per cent.

Albarran.....	3.5 per cent.
Rovsing.....	3.3 " "
Boeckel.....	5.8 " "
Wildbolz.....	4.8 " "
Frank.....	9.3 " "
Legnen and Chevasser.....	5.9 " "

I. *Ultimate result of nephrectomy:*

Kümmel.....	32 of 43 patients living and well.
Albarran.....	34 of 39 patients living and well.
Kelly.....	Complete cure in 21 where bladder not involved and in 18 of 36 where bladder was extensively diseased.
Israel.....	11 of 29 cured.
Küster.....	11 of 17 cured.
Schede.....	16 of 22 cured.
Czerny.....	11 of 27 cured (secondary nephrectomy done in 16).

The primary lesion is always a miliary tubercle. These tubercles, as they increase in number, tend to coalesce, become mixed, infected, and form pus cavities of varying size in the kidney parenchyma. The process usually begins at one pole of the kidney, commonly the lower, and as the abscess cavities form and increase in size or number, the kidney tissue is destroyed, the viscus finally being transformed or converted into a series of pus-pockets or caverns, the ulcerocavernous form of kidney tuberculosis.

These pockets may communicate with each other. They are filled with mushy, yellowish, necrotic material. The pus-cavities are usually lined by a smooth wall, or if the process of destruction has been a rapid one, the wall consists of granulation tissue. The kidney itself may be enlarged, soft, or even fluctuating, and tender to touch and more or less nodular. In the later stages it may be shrunken in size or very large—hydronephrotic. Finally the disease extends to the pelvis: it becomes dilated and filled with pus. Next the ureter is involved; and if, as the result of the inflammatory process, its lumen is occluded, the kidney becomes enormously enlarged, the result of the retention of the disease-products as well as of such urine as this diseased kidney is still capable of excreting.

Taking tuberculosis of the lung as a model, any man who is keenly interested in tuberculosis should study two lesions: one that he sees on the surface in the shape of a lupus, and the other, which he can find any day in the necropsy room of any large hospital, the lesion in the lung. If you will study these two lesions as models and observe the destruction and repair of tissues, you will have a fairly good mental picture of what the lesion can be when it is situated in other organs or tissues in the body, except as influenced by a mixed infection and influenced by local cellular conditions.

In my very earliest experience with tuberculosis I began to look on all tuberculous processes as I would on a lupus, and I brought a picture of the organ involved metaphorically to the surface and studied it from an external view standpoint. That is the only way to acquire an accurate idea of the resistance, repair, and destruction of tuberculosis. We are not told in the history what was the general constitutional condition of this patient before the beginning of the local symptoms. You may err in the early diagnosis of tuberculosis of the kidney unless you take into consideration the constitutional symptoms that preceded the local manifestations. They are, a slight afternoon or evening elevation of temperature, increasing fatigue, a coated tongue, and an inability to do the work with the same ease and facility that

the patient did before. The diagnosis made almost uniformly in the primary stage of all the tuberculous lesions is malaria.

Now, coming to the local symptoms, the first subjective symptom to direct the patient's attention specifically to the kidney is the pain. It is commonly the initial symptom that really alarms him. The urination symptoms, however, are really the primary ones. Frequent urination is the most common early symptom of tuberculosis in the urinary tract. In the case of kidney tuberculosis pain exists only on the diseased side, and is more marked at night. Instead of a polyuria, there is at first commonly frequent micturition and no increase in the quantity of urine. Then comes the polyuria, an increase in the quantity of urine, and the frequent inclination to urinate. There is something about the desire to urinate that is characteristic of kidney, bladder, and epididymis tuberculosis, namely, that when the patient has the inclination to urinate, he has to go at once or he is unable to hold the urine. Many times, even in the very early stages, they have to wear a urinal to prevent soiling the clothing. This is so uniform an occurrence that it should be just like a red flag signal of the disease; when the patient complains of this urgent inclination, it should direct one's attention to the danger to life—the kidney in this case.

Later comes the incontinence. It may be present in the daytime or at night; he may urinate in his sleep without awakening, and the incontinence may come as a sudden impulse in the daytime. Next comes the tenesmus, the cramp in the cervix or bas-fond of the bladder often preceding, occasionally occurring during or even after, urination, and the patient may have a prolonged burning sensation in the bladder after urination.

Finally, there is the hematuria. Hematuria may be the first real manifestation of a kidney tuberculosis next to the constitutional symptoms. The patient may suddenly be taken with a hematuria while at work; or on awakening some morning or on retiring at night he has a sudden flood of blood in the urine. Then, when you examine this class, you find no physical signs of tuberculosis, but the tuberculin test would show a positive reaction.

The discovery of tubercle bacilli in the urine, with pus or blood, is always a positive evidence of kidney or bladder tuberculosis, but often a late sign: when the ureter is blocked, this evidence is lacking. It is always essential to localize the disease to the kidney and to ascertain which kidney is infected, and it is usually a unilateral affection, because in most cases the infection is a hematogenous or embolic one.

The fact that the disease is very amenable to treatment if recognized early makes an early diagnosis absolutely essential. The diagnosis can, as a rule, be made easily when the disease is advanced, but it is then rather late to expect much from any form of treatment. Therefore, the time to make the diagnosis is before the bladder becomes involved, before the appearance of constitutional symptoms, of sepsis, and while the disease is still confined to a small area in the kidney.

The diagnosis should be based on the general appearance of the patient, the increased frequency of micturition, with or without an increase in the quantity of urine voided, the presence of pain, dysuria, tuberculin test, polyuria, hematuria, the bacteriologic examination of the urine, and cystoscopy. The study of the urine, however, is most important. The cystoscope and the ureteral catheter have revolutionized the diagnosis of kidney diseases. A cloudy or bloody urine coming from one ureter often settles the diagnosis, but even cystoscopy may be misleading and give no information. It is only by assembling all the evidence received from the various aids to diagnosis that a correct conclusion can be arrived at. The kidney function tests are also of value in determining the advisability of a nephrectomy, but we must remember that a tuberculous kidney in the early stages of the disease may functionate normally.

This young man, as you see, went along for a number of months with various diagnoses, gastric ulcer, etc. He was not operated on. He came to us for operation, but we did not operate, because he was then in rather a bad condition generally, and the question was whether we should do an immediate operation for the removal of the kidney or whether we should increase his



Fig. 119.—Shows the tuberculous pathologic process originated in the pelvis, and slowly, by continuity of ulceration, invaded the cortex and extended into the ureter.*

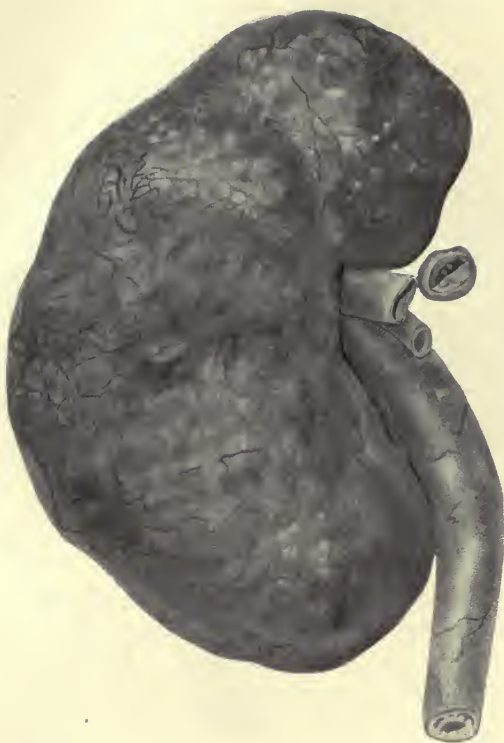


Fig. 120.—The same kidney as in Fig. 119, showing the external surface with capsule removed. Note the vascularization on the surface, also a small stone found in the pelvis, which was probably the focalizing cause of the tuberculosis.



Fig. 121.—Shows the cortex involved with miliary tubercles, which have extended to the capsule. No material enlargement of the kidney and no infection of the pelvis or ureter.

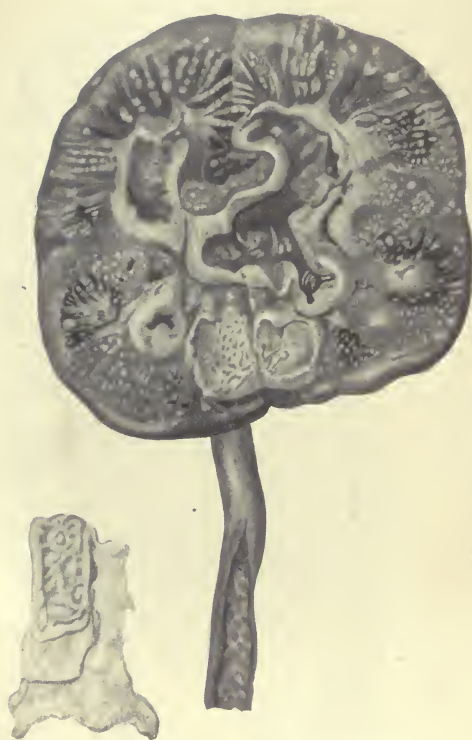


Fig. 122.—Showing a combination of cortical pyramidal pelvic and ureteral tuberculosis.



Fig. 123.—Showing almost complete destruction of the kidney from tuberculosis.
The primary focus cannot now be determined.

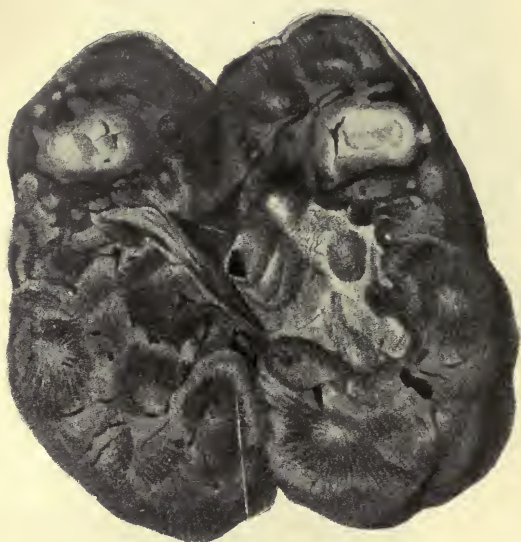


Fig. 124.—Showing only the upper pole tuberculous, but the pockets communicated with the pelvis, which became infected; the ureter was also ulcerated, as shown above.

resistance and endeavor to get him in better shape for the operation.

The question whether one should or should not operate in renal tuberculosis is based with us on two factors: first, the degree of destruction of the kidney as governing the possibility of repair, and, second, the degree of obstruction of the outlet—the ureter. The degree of destruction, that is, the stage to which the pathology has advanced to at the time, is an important factor in so far as one is able to determine from the physical signs.

If patients, in the early stage of tuberculosis of the kidney, are put under good hygienic and properly administered tuberculin treatment, so there can be a complete repair of the disease and for an indefinite period of time, they may become perfectly well. There is no question about that, as it has been demonstrated repeatedly in our experience. Then, again, there are patients who have advanced lesions, with little resistance, and unless you operate early, the patient will succumb from a general tuberculosis. So you can see that you have to take into consideration the element of the patient's resistance, and the fight which his tissues are putting up against the disease. The location of the primary focus of disease in the kidney, that is, the point at which the lesion begins in the kidney, has much to do with the result. The lesion begins in the kidney in three different positions: First, in the subcortical zone; second, near the apex of the papillæ; third, in the pelvis of the kidney. These are the three positions of primary focal invasion. If you put them in the order of frequency of occurrence, then you would say that the most common was the papillæ lesion. The cortical tuberculosis offers the best prognosis; the papillary lesion repairs itself next in frequency, and the pelvic tuberculosis is the most unfavorable. The accompanying pictures illustrate beautifully the most common locations and methods of destruction of the kidney. In the advancement of the disease from the papillæ into the pelvis there is not uncommonly associated a mixed infection, coming either from below, usually the bladder, or by way of the blood-vessels. A mixed infection with tuberculosis, you know, is the most serious of all conditions that occur in connection with tubercu-

losis in any organ. Practically in all tuberculous lesions tuberculosis never kills of itself. It kills as a mixed infection.

The next point I want to mention, and with a good deal of force, is the presence of obstruction to the outlet of the kidney or an obstruction to drainage—an obstruction to the ureter. With this obstruction the patients have recurrent chills and fever, rapid emaciation, intense septic intoxication, and an early fatal termination. If you have good drainage, tuberculin offers a favorable prognosis.

As the patient is ready for operation, we will defer further discussion until later.

We will expose the kidney from behind by the lumbar route, making a subcostal curved incision, coming well forward toward the outer margin of the rectus muscle. Avoid the iliohypogastric nerve. It is readily recognized beneath the muscles. The patient is spare, and therefore the kidney at once comes into view. It is coming easily into the field. The capsule is firmly fixed to the kidney by cicatricial tissue. I am going to bring up that kidney and look it over and decide on what I will do. I wish to do as little pressing and as little handling of that kidney as I possibly can, therefore I am going to try and free it entirely of its fatty capsule before I bring it up into the field. There is the kidney! Let us look it over. I can feel a mass in the upper pole as of resistant nodules. There is a nodule. That feels hard enough to be a stone, although the skiagram was negative. He has tubercle bacilli in the urine, but so far as we were able to determine, he has no tuberculosis in his lung. I did not go into the analysis of the frequency of the presence of tubercle bacilli in the urine of patients suffering from pulmonary tuberculosis. Remember that they may have tubercle bacilli in the urine without a focal tuberculosis in the kidney and then, again, there may not be any present.

There is an apparently thickened ureter. Yes, on palpation, it is pronounced. All the kidney is thickened. Now I have the renal vessels exposed. I do not wish to open the pelvis of that kidney now because if I do open it I run great risk of infecting the field. I will free the ureter and the pelvis of the kidney en-

tirely from the vessels before opening the pelvis, thus lessening the likelihood of an infection. Now we have all these structures separated, and there is not the slightest risk of an infection of that zone.

We are ready to remove the kidney. We will clamp the pedicle—vessels, ureter, and all—with our special clamp, which I have designed for this purpose, placing two of them, one from

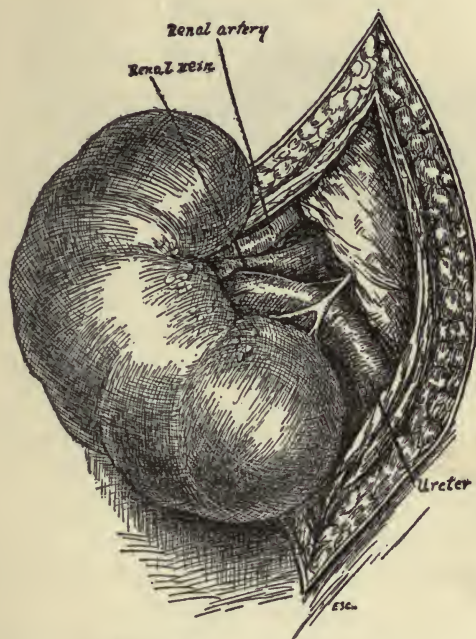


Fig. 125.—Nephrectomy. The kidney has been brought out onto the surface so as to expose the renal vessels and ureter, the latter being greatly dilated.

the upper and the other from the lower pole directions. The handles of these clamps are removable, so that the clamps are left in place until such a time as it is considered safe to remove them, usually three to five days. The continued closure is assured by means of a strip of gauze which is tied securely around the end of each clamp as it protrudes from the wound. If I were not going to leave the clamp on permanently, I would divide the ureter, as illustrated in Fig. 127, tie the vessels, as illustrated in the

same figure, invert the end of the ureter, as illustrated in Fig. 128, and close the wound completely without drainage. The pedicle is divided above the clamp.

Now let us bisect this kidney. There is a stone in one of the upper calices, as I described it on palpation, but it did not show in the skiagram. It is really a calcified mass of tissue. There

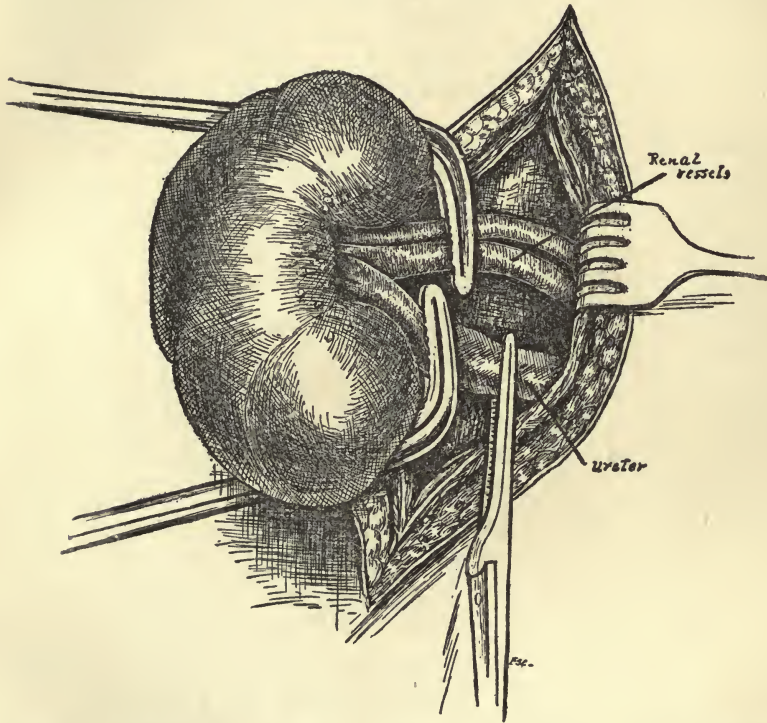


Fig. 126.—Nephrectomy. The renal vessels and the ureter have been clamped separately with Dr. Murphy's special kidney pedicle clamp.

are many pus-cavities or cysts filled with calcareous brie. It looks like a calcareous rather than a tuberculous brie. That makes it right for taking out the kidney, and it shows the great repair that has taken place in the tuberculous process, as the tissue around the stone looks more like a connective-tissue inflammatory mass than a tuberculous granulation. There is some

apparently healthy kidney tissue in the lower pole. It was in the calices of the upper portion that we found the stone embedded, the most common position for a tuberculosis.

We will cauterize the ureteral pedicle thoroughly with 95 per

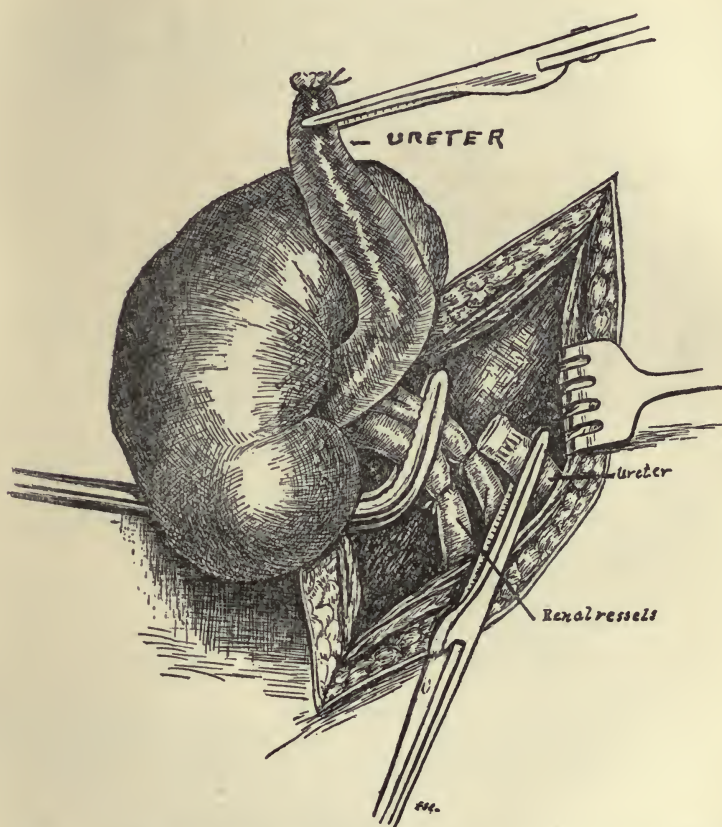


Fig. 127.—The ureter has been divided between forceps, the distal end ligated to prevent the soiling of the field by the outpouring of fluids from the kidney. The renal vein and artery have been ligated separately, but not yet divided.

cent. phenol and not neutralize it. There is not the slightest oozing anywhere. We are now ready for the closure of the wound, which is accomplished with figure-of-8 silkworm-gut stitches, which include all the structures of the abdominal wall. There

is the iliohypogastric nerve. See it right there in the field. We do not wish to include that in the suture; if you do, it gives the patient very much pain following the operation. The skin-edges will be approximated with horsehair; no drain. A large dressing of plain sterile gauze is applied.

In regard to the management of this stump, remember we cauterized it with 95 per cent. phenol without neutralizing it. There is an accurate apposition of this tissue immediately, so we

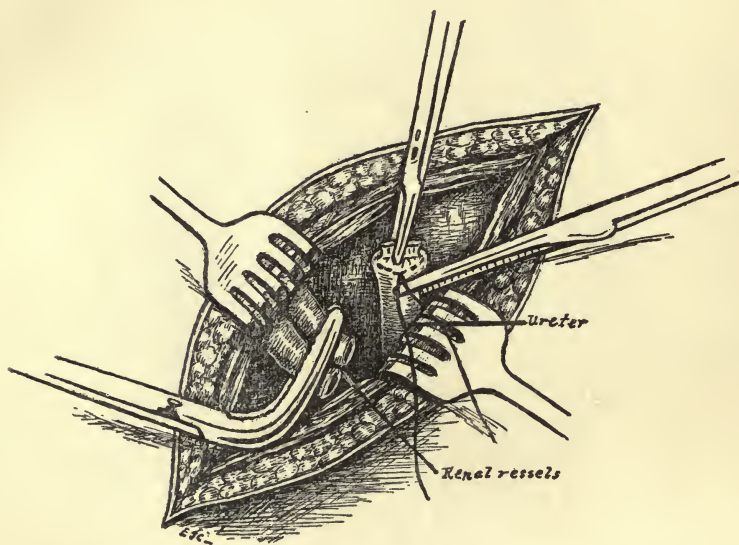


Fig. 128.—The renal vessels are divided and the purse-string suture has been placed on the proximal end of the ureter, previous to its inversion, as in the case of the appendix. The kidney has been removed.

feel it needs no irrigation or foreign substance stimulation. We use the figure-of-8 silkworm-gut stitches in these kidney cases, just the same as we do in closing the anterior abdominal wall, approximating all the structures completely, so that we will not have a hernia subsequently.

The pathologist reports numerous small tuberculous foci with giant-cells and tubercle bacilli. The tuberculous process was being rapidly encapsulated by connective tissue. Round-cell

infiltration in many areas, with small foci of suppuration. Round-cell infiltration of the pelvic mucosa.

The clamps were removed on the third day; complete primary union followed; uninterrupted recovery. There were no tubercle bacilli found in the urine on repeated examinations following operation, but notwithstanding this the tuberculin treatment will be continued for many months.

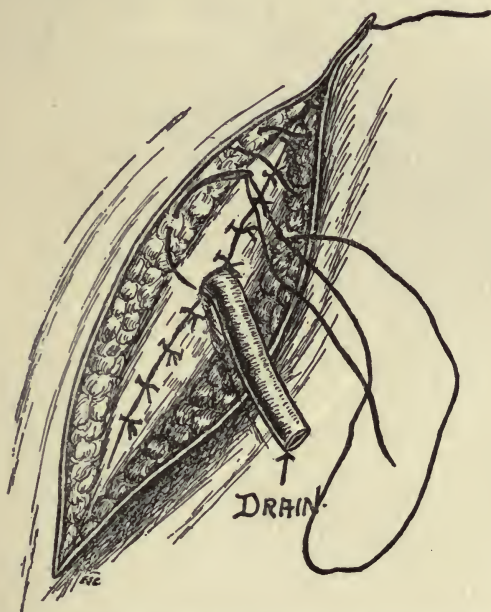


Fig. 129.—The ureter and renal vessels have been dropped back into the wound, the rubber tube drain placed, and the muscular layers of the wall closed with continuous catgut sutures. The continuous catgut stitch is being placed subdermally. Greatest care should be exercised to avoid soiling of wound.

The prognoses in operation mortality, as well as the ultimate results, are shown in the synopsis. I have two patients—one twenty-three and one twenty-one years—after nephrectomy for tuberculosis living and well.

The next case is somewhat similar to this one, and we will take it up now to emphasize the points we have made. Listen to her history:

HISTORY OF CASE II

CASE II.—Patient, Mrs. J. R. female, aged twenty-seven, married, entered the hospital the first time October 28, 1913, because of intermittent sharp pains under the right costal margin and pains in the bladder region, which occur usually only when the bladder is full and during urination. She urinates frequently, passing small quantities at each urination; gets up three or four times during the night. There is no incontinence or dribbling, and the urine has never contained blood or pus, so far as she knows.

Present trouble dates back to December, 1910, the day after her last childbirth, when she began to have sharp pains in the lower pelvic region, but no chill or elevation of temperature. She tried to urinate, but could not. Looking at her abdomen she noticed a moderate, rounded swelling above the symphysis pubis, which was rather firm and tender to touch—evidently the bladder. On this day she was catheterized three times by the nurse—the first gave a large quantity of urine. For the next nine days she was catheterized a number of times each day. After this she could urinate, *but had severe pain during the act*, but preceding the catheterizations she had had no pain. Urination was increased in frequency (about every hour at night), but without any diminution or increase in the total amount of urine, nor did it contain either blood or pus.

This condition continued throughout the next year, during which time she took electric treatments for the first two months and internal medication the remainder of the time. The frequency of urination diminished, and the pain gradually became less severe, but did not cease entirely.

In September, 1912, she began to have severe intermittent pains again under the right costal margin, usually persisting for three or four hours. She has had four attacks of this subcostal pain since then. The bladder symptoms have been less severe, and she does not have to get up so often at night to urinate. In July, 1913, during one of the attacks (it was the last one), she passed urine which she thinks contained some blood. She has

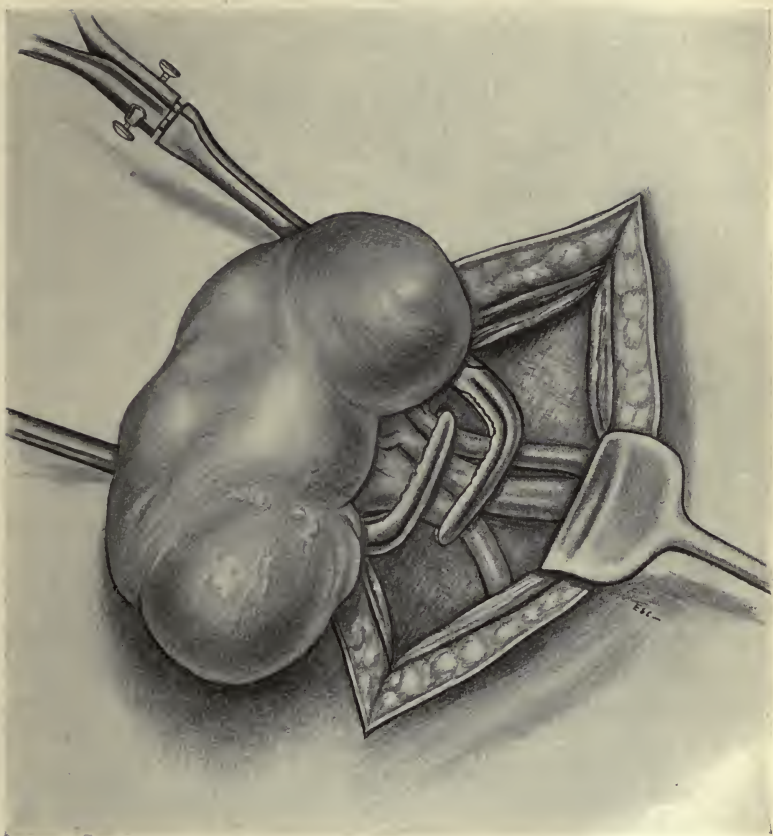


Fig. 130.—Nephrectomy. In this case the renal vessels and ureter were clamped together, the pedicle being divided between the two clamps.

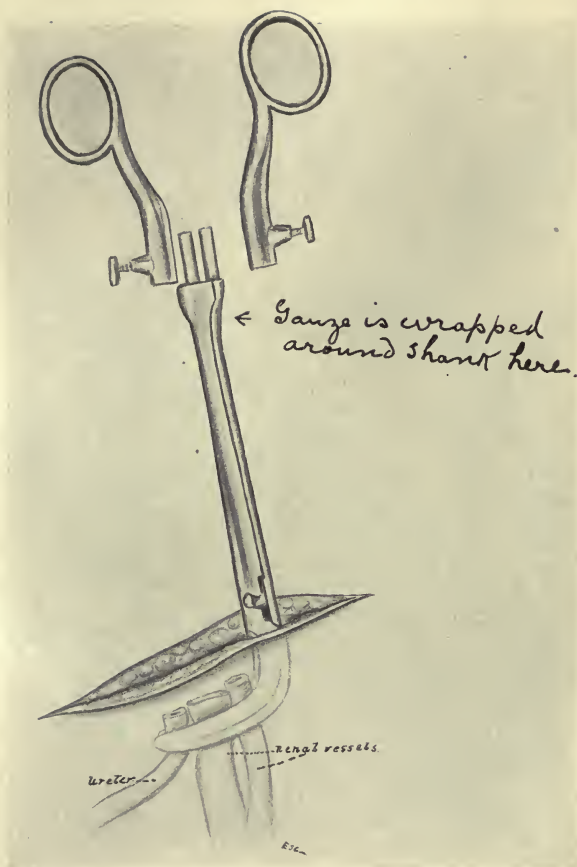


Fig. 131.—Nephrectomy. Only one clamp was left on the pedicle in this case, and it was not removed until the fifth day. The handles of the clamp are always detached, and the shank is tied with gauze to insure the locking of the clamp. The wound is closed up to the clamp.

never had incontinence or dribbling, or suppression of urine. She has gained in weight since the onset of this trouble. She has no pain in the left side of the abdomen. Her appetite is good; bowels are regular, and she has never had cough or night-sweats.

At this time the urine was injected into a guinea-pig and after six weeks her doctor reported this test positive for tuberculosis. In October, 1913, many tubercle bacilli were found in the urine of a non-catheterized specimen, a direct smear having been made.

Menstrual history: Began at twelve years, always irregular, very painful, lasting four days, three or four napkins daily; no clots; no leukorrhea. Since the birth of her baby menstruation has been more regular and less painful. She has had only the one child; no miscarriages. Last menstruation was October 16, 1913; lasted four days, three or four napkins a day; no clots.

Previous history: She had typhoid-pneumonia at the age of eight; measles at fifteen; scarlet fever four years ago. When three months pregnant she was thrown from a buggy and landed on her right side. She had pain in her right hip for a week.

Family history: An aunt died of "tuberculosis of the throat." No history of carcinoma in the family.

Cystoscopic examination by Dr. Murphy October 28, 1913, showed no congestion or swelling of the left ureteral opening, which was easily brought into the field; a retraction of the right ureteral opening. The left ureter was catheterized, and the urine was found normal, only an occasional pus-cell and a few red cells appearing in the specimen. The right ureter could not be catheterized, because of the retraction of the opening and obstruction three-fourths inch up. The amount of urine, however, which passed from the right kidney into the bladder was only one-fourth the amount which passed by the way of the left ureter, and contained many pus-cells; several examinations showed 20 to 40 to the one-sixth field. Renal function test (phenolsulphonephthalein) showed 51 per cent. for the first and 12 per cent. for the second hour, indicating that the excretory function was fully up to normal.

Physical examination disclosed an enlarged right kidney. A

hypodermic tuberculin test (5 m, dilution No. 2, Koch's old tuberculin, made by Lucius and Bruning) was made and proved positive. Diagnosis, tuberculosis of the right kidney, and its removal advised.

The patient did not remain in the hospital at this time, but returned for operation December 27, 1913.

The blood count made on January 1, 1914, showed 9400 leukocytes.

Urine examination on December 31, 1913, showed a specific gravity of 1018, acid reaction, no albumin, no sugar, 10 to 12 white cells to the one-sixth field, few epithelial cells. Urine examination on January 1st showed a specific gravity of 1019, acid reaction, no albumin, no sugar, 20 to 25 white cells to the one-sixth field; an occasional epithelial cell.

COMMENTS AND OPERATION—CASE II

DR. MURPHY (January 3, 1914): That gives you a fairly good history of this case. First she gave birth to a child; second, following that she was catheterized; third, following the catheterization, she had an infection; fourth, following that infection she has had pus in the urine continuously. Then came the pain in the right upper abdomen—that was not a colic; then came the enlargement of the kidney; then the recurrence of the pain with colic, and then the obstruction to the outlet of the kidney. Then came the long period of malaise and lassitude during the acute destructive stage of the tuberculosis.

She came here first about two months ago with a tumor in the right kidney region. Then we made some tests and they showed, first, that she had pus in large quantities in the whole specimen of urine; second, when we catheterized both ureters, no pus came from the left ureter, but a large quantity of pus was found in the urine coming from the right kidney, and when we made the efficiency test of the kidneys, that is, the phthalein test, it showed that she had three times as much urine coming from the left kidney as from the right, which means that only one-fourth of the work, as far as quantity is concerned, was being done by the right kidney, and very much less than one-fourth of

the work, as far as urea is concerned, was from that source. That gives us the justification for the removal of this kidney. We have cleared up the fact that the left kidney is not involved, and that the right kidney is doing little or no work. The tuberculin test which we made when she first came here was positive, and the guinea-pig test made previously was also positive for tuberculosis. In addition to all these findings, when she first came in, the catheterized specimen of urine from the right ureter contained many tubercle bacilli, showing that it was not only a tuberculosis of the right kidney, but an active tuberculosis. As long as that is going on there is danger to the patient. She also has a large tumor in the right side, showing there have been retention in the pelvis, distention, and probable ulceration and destruction of the cortical portion.

We have settled the fact that as soon as a tuberculous kidney is removed these patients get well. We have also settled the fact that where one kidney is doing a large part of the work, putting the additional percentage of the work on that kidney by removing the diseased one should not overtax the well kidney. Remember, the splendid work written by Morris, of Scotland, in which he showed that when the kidney that was practically entirely destroyed in its secreting potency was removed, the mortality was only 3 per cent., while the removal of a kidney that was approximating 100 per cent. normal function gave a mortality of 30 per cent. or more. Removing a kidney is not merely the successful taking out of the diseased organ: it is the taking it out at a time when the other kidney has adopted or is capable of assuming the whole renal function.

In this case we will make an almost transverse incision just below the margin of the ribs, because we must have considerable space here on account of the size of the kidney. There is the kidney, right in the field, just to the outer side. There is the pleura. You can see it moving up and down; the pleura on this side does not come down as low as it does on the left side. We must keep that in view so as not to divide it. (We lost a splendid dog specimen the other day from opening the pleura on the left side in doing some additional experimental work on the

kidney.) That is the fibrous portion outside of the fatty capsule. There is the kidney itself, shining through with considerable vascularization around it and onto its capsule. There is a fibrous adhesion of the capsule to the surface of the kidney, showing that there was a mixed infection in this neighborhood. I am trying to get that free without opening the peritoneum or the kidney. There is the iliohypogastric nerve, which we wish to avoid. Now we will try and bring the kidney into the field without injuring the neighboring tissues. We are just behind the intestines here.

Now we have the kidney fixed at its upper pole. There are some vessels that have developed across and through the kidney. That is an extravascularization, not a normal vascularization at all. We will free the lower pole to see if we have any collateral circulation here. There is a small vessel coming out above, out of the regular position. Now we are coming onto the renal vessels. See! there are the renal vein and artery and the ureter. We will not cut that ureter, because the treatment or management of it is the all-important thing for a primary healing after nephrectomy for a tuberculosis. The pelvis is dilated from the accumulation of pus within it. The ureter is also dilated and infiltrated. It is three times the normal size.

Now we will ligate the vein and artery separately, putting two ligatures on each. We divide each vessel above the highest ligature, between it and a large hemostat, which was placed in position to prevent the flow of blood and other fluids from the kidney into the wound.

A hemostat is also placed on the ureter, which latter is divided below the hemostat, the areolar tissue wall of the cut end being caught with other hemostats as we proceed with the division. (See Fig. 127.) I want to turn in these cut edges, make an entropion, so that no infective material can get either in or out. The care of that ureteral stump is the all-important proposition.

I do not know that I can infold it, but I think it is possible. I do that with fine chromicized catgut, as one turns in the stump of the appendix, and I have succeeded. (See Fig. 128.)

In every step of that procedure we were absolute masters of

the whole situation. We put two ligatures on the vessels, following the plan outlined by Senn in the splendid experimental work he did on the arteries so many years ago. There is the fatty portion of the capsule now, which we will fix around this edge clear up to the surface, so as to keep that away from the infected zone; the next step is to restore the musculature of the abdominal wall, so that we have the full strength of that wall. Transversely cut muscles, you understand, when they are divided and united end to end, give fully as good a support as the original muscles if the nerve supply has not been disturbed.

The technic of this operation is not difficult if one is patient in the freeing of the kidney from the intestines, otherwise you can get into very serious complications. Here we had only a moderate dilatation of the pelvis, consequently we had no difficulty in getting the pelvis free; but where you have a great dilatation, as occurs in some of the cases, you have to be very careful in taking out the kidney. Now we have sutured all these muscles back in their normal relationship with catgut, and now we are putting in the figure-of-8 silkworm-gut stitches. The edges of the skin incision are approximated with horsehair. No drain.

Let the record show that the kidney was exposed through a long subcostal incision, which gave room for the removal, leaving the true capsule on the kidney without rupture after bringing it well to the surface.

There is one point which I wish to make in connection with great dilatation of the pelvis and sacculated kidney; that is, that the sacculation occasionally extends inward behind the renal vessels to the vena cava. In removing one of these, as we endeavored to free the sac from behind we came into the vena cava, which we recognized; the sac was firmly adherent to it. In the separation we tore an opening in the vena cava. This rent we clamped with 3 or 4 hemostats and continued the separation until it was freed from the under surface of the renal vessels. We then, with a conjunctiva needle and fine silk, sutured the opening in the vena cava. This accident fortunately occurred after we had done our experimental work on arteries and veins, making us feel at ease. Bear in mind that the blood in the vena

cava has scarcely a plus pressure and the finer stitch prevents the bleeding. The patient made an uneventful recovery. In the sacculated kidney cases without infection and with normal secreting function we have a mortality of 90 per cent. in the removal; you cannot take out the kidney without great danger to the patient's life on account of throwing all of the work suddenly on the other kidney.

In managing that class of work you have no trouble at all. You can cut off the sac, then split the ureter, and unite it to the edges of the amputated stump of the pelvis—just sew it on like a patch on a pair of trousers. There is no danger whatever of a non-union. The ureter remains united. You can sew it in any position even with the urine flowing around it and you get a very rapid union. The reason for that is its great vascularity. Here there was no indication for doing anything but a radical removal of the kidney on account of the diagnosis. There was no occasion for resecting any portion of the kidney as might be done when the tuberculosis is circumscribed. The moment you rupture the pelvis of a tubercular kidney, you hazard the result; that is, you get a tuberculosis in the wound pocket and that tuberculous infection takes months and months to heal. Where we put on the ligatures we endeavor to crush out the mucous coat of the ureter, so we will have connective tissue approximated for union. If you crush with the clamp, then you have two connective-tissue surfaces in apposition where you tie the ligature, in place of two mucous surfaces which have been crushed out. The statistics of the nephrectomy are given on the board. That table gives a fairly good synopsis of the results, primary and remote, from operation on the kidney.

HISTORY OF CASE III

CASE III.—Patient, Mr. V., aged nineteen, entered the hospital February 9, 1914, and gave the following history:

Family history is negative to tuberculosis and carcinoma. Venereal history is negative.

Past history: Had measles at nine and chicken-pox at fourteen years of age. No complications followed either.

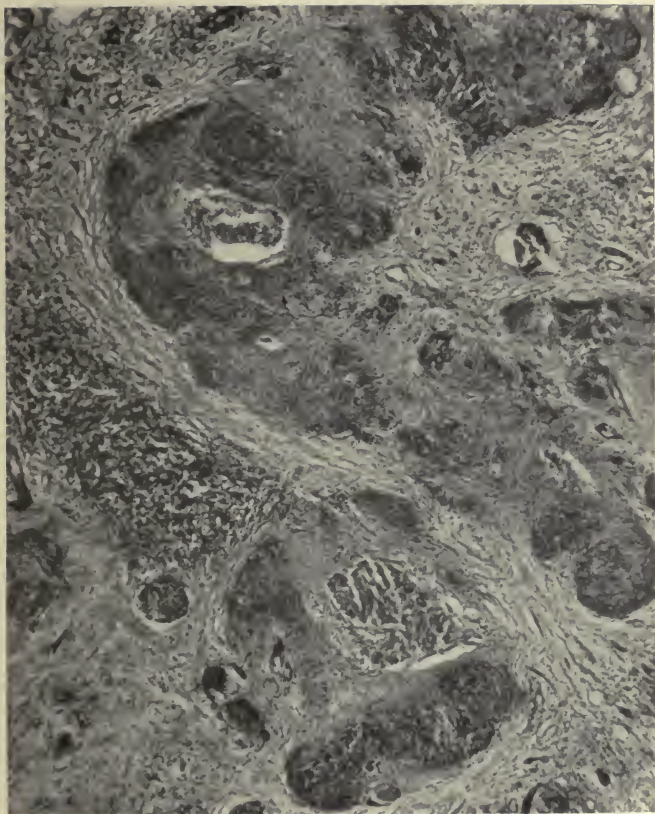


Fig. 132.—Tuberculosis of kidney. Section through cortex of kidney, showing tubercles in various stages of development and formation of much dense fibrous tissue.



Fig. 133.—Tuberculosis of kidney—ulcerocavernous type. The pelvis was distended with pus; many miliary tubercles in cortical zone, which was very thin, with abscesses in the pyramidal zone formed by the coalescence of ulcerating tubercles. In the lower pole the secreting tissue was almost entirely destroyed. The disease began in pyramidal zone, and not in the pelvis.

Present illness: During July and August he tired easily—felt weak. About September 15, 1911, he had a gradual onset of frequent urination; at first he had to get up once and later two and three times each night. He urinated four to six times a day. This gradually increased as time went on, and with the increased frequency he had dull, aching pain in the left ilio-lumbar region and a tired feeling after each day's work. This condition continued until October 1, 1913, when he began to have sharp, burning pains at the end of the urethra at every urination, which condition has continued up to the present time.

About November 20, 1913, he first noticed blood at the end of each urination, usually in clots; this continued until the clots finally disappeared in about three months, at which time the urine first passed was clear and gradually became blood tinged, and before the completion of urination he passed almost pure blood. He has had a daily elevation of temperature of about one degree for the past year. He never had chills until January 5, 1913, when he was given a tuberculin test. That night he had a severe chill, lasting one hour, and a high temperature the next day. A pronounced positive reaction. Following this all the blood disappeared from the urine for one day and his general condition was improved for a few days. He always has severe pain on urination after eating such fruit as oranges or lemons. At present he is tender over the left kidney region. He has lost about ten pounds in weight during the past year. His appetite is fair. His bowels are constipated. A tumor could be felt under the right costal arch.

CYSTOSCOPIC EXAMINATION (BY DRS. KREUSCHER AND CAREY)

Bladder mucosa inflamed. Opening of right ureter normal; not catheterized. Opening of left ureter red and elevated. Ureteral catheter could be introduced only three-fourths inch. The urine from the left ureteral opening was bloody and small in quantity.

Urine examination: February 10, 1914, bloody appearance; specific gravity 1018; acid in reaction; albumin; white and red blood-cells in abundance; no casts.

The leukocyte count on February 9 was 7000. Phthalein test showed 48 per cent. first hour and 18 per cent. second hour. Diagnosis, tuberculosis of left kidney.

Pathology.—This kidney (left) was removed February 11, 1914, through a subcostal incision in the usual manner, one clamp being placed on the pedicle. This clamp was easily removed on the fourth day, after the operation, without any bleeding resulting. Primary union followed. Tuberculin will be continued for months.

The kidney was dark red in color, nodular, and slightly enlarged. The pelvis was dilated. The capsule peeled off quite easily, and the surface of the kidney was studded with pin-head-sized yellowish masses. (See Fig. 121.) On section the pelvis was greatly dilated; the pyramidal zone consisted of a number of abscess cavities, some of which were intercommunicating. The cortical zone was thinned out and studded with miliary tubercles. The abscesses and kidney pelvis contained a typical tuberculous brie (type illustrated in Fig. 122).

[Primary union followed removal of the clamps.—ED.]

VESICAL PAPILLOMATA

A TALK BY DR. LEWIS WINE BREMERMAN AT THE
CLINICS, NOVEMBER 29, 1913

DR. MURPHY: While I am closing up this case, Dr. Bremerman will speak to you about a papilloma of the bladder which he is about to remove.

DR. BREMERMAN: *Gentlemen:* The case which I will present to you this morning is one of recurrent papilloma of the bladder. Before going into the history of this case let us consider for a few minutes some of the more important features of vesical papillomata.

A few years ago vesical papillomata were considered a rather rare condition, but at present, with our better methods of diagnosis, the urologist is coming in contact with these cases quite frequently, yet fortunately papillomata form only a small percentage of bladder affections, consisting of about 3 to 5 per cent. of all genito-urinary operative surgical conditions.

It is estimated by various observers that vesical tumors form only one-quarter to one-half of 1 per cent. of all tumors, but if our recent statistical estimate be correct, the bladder as a seat for this pathologic condition is of more frequent occurrence than was generally conceded.

Statistics computed by various authorities show that papillomata are of much more frequent occurrence among males than among females, in the proportion approximately of about three to one. It is difficult to obtain accurate data upon this subject, due to the fact that the special work of the various investigators must be considered. As a rule, the urologist sees more men than women; with the gynecologist the reverse is the case. In my work the male cases are in proportion of two to one over the females.

The ages of the individual suffering with this condition vary.

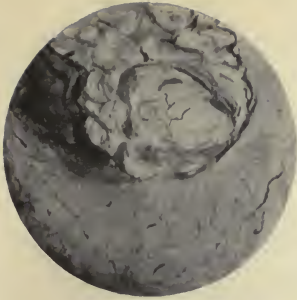
Some cases have been reported as young as sixteen years and as old as seventy-five. In the cases which I have seen the youngest, a male, was twenty-four years of age, the oldest, a female, was sixty-eight, with an average of forty-five years; therefore, it may be assumed that vesical papillomata are associated almost entirely with adult life.

The direct cause of tumor of the bladder, as with tumors elsewhere, is absolutely unknown. Many theories have been advanced to account for these tumors, but they are all practically exploded. It has been considered that, owing to the frequency of certain cell inclusions which have occurred in the epithelial variety of bladder tumors, certain parasitic elements were responsible to a degree, at least, for their origin, yet the weight of evidence is against such a theory, for the actual organism has never been isolated or demonstrated.

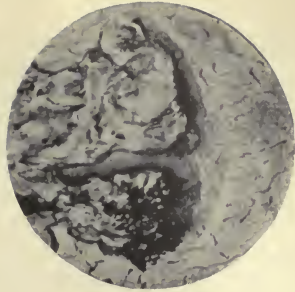
Personally, I feel that the Cohnheim theory explains, at least with a reasonable degree of certainty, the theory of the development of all tumors, and, until more substantial evidence is reported, holds good in tumors of the bladder.

In all my cases there has been a long-standing inflammatory condition of the bladder, which has persisted in some cases under careful active treatment, but in most cases a very inadequate form of treatment has been employed. As far as occupation is concerned, it need not be taken into consideration, as I am sure it plays no part as an etiologic factor. I could trace no connection in any of the cases where a chemical irritation might be responsible for the condition, as so reported by some observers. Mechanical irritations of the bladder of long standing, produced by foreign bodies, such as stone, etc., are considered by some authorities to bear a very definite relation to the formation of tumors; others, however, are of the opposite opinion.

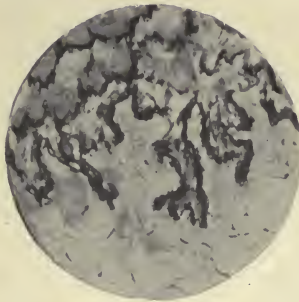
In the cases I have seen I have never found a distinct calculus. There have, however, been some crystalline incrustations noted, which is not at all unexpected. I have never seen a tumor in any of my stone cases, with the exception of one where there had been two suprapubic cystotomies performed, one for stone, the other for a prostatectomy. I removed a stone some months



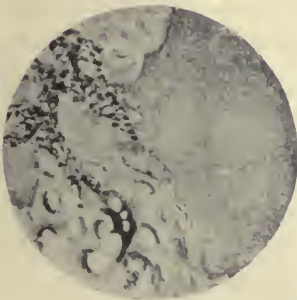
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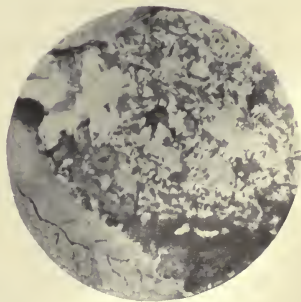
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Fig. 134.—1, Single pedunculated papilloma, left side, posterior wall; 2, single pedunculated papilloma, bifurcated; bleeding point evident; 3, multiple papillomata from anterior bladder-wall; 4, carcinomatous recurrence, posterior wall of bladder; 5, primary carcinoma of prostate and bladder.

after the prostatectomy, and there was evidence of some papillomatous tissue, which proved to be typical papilloma.

Tumors of the bladder are variously classified, but the classification of Küster and Davis seems to be the most practical:

I. Epithelial Group:

- (a) Papilloma.
- (b) Carcinoma.
- (c) Adenoma.
- (d) Cysts.

II. Connective-tissue Group:

- (a) Sarcoma.
- (b) Myxoma.
- (c) Fibroma.
- (d) Angioma.

III. Muscular Group:

- Myoma.

The papillomata are by far the most frequent in occurrence, and develop in the mucosa and submucosa. Histologically, these tumors are composed of a framework of connective tissue, with a network of fine blood-vessels completely covered with epithelial layers, usually pedunculated, yet they may develop over quite an area, or several may arise from a single pedicle. The network of blood-vessels accounts for the frequent hematuria associated with these cases, due to a breakdown of the epithelial layers, resulting in ulceration and destruction of the surface, exposing the vessels, proving that the development of the growth has progressed to a marked degree. I am of the opinion that if the diagnosis be made early, prior to the appearance of a hematuria, there would be fewer cases of recurrence. Unfortunately, the cases do not come to the urologist direct—they usually go through a long period of treatment before coming under the observation of the specialist whose cystoscopic skill is such that an easy diagnosis may be made. The diagnosis lies entirely in the use of the cystoscope, so that the character, location, size, and the general appearance of the bladder may be thoroughly studied, and from the picture definite measures may be outlined as regards treatment.

A small portion of the growth may be procured by means of the cystoscope, and a microscopic examination made from a section, affirming or contradicting the diagnosis, together with a definite knowledge of the variety of tumor with which one must deal.

It has been proved that vesical papillomata are exceedingly prone to recur, yet, as a rule, the recurrence is not apt to be at the site of the primary growth; it is usually outside of the previous operative field. The recurrence occasionally is evidenced in the scar of operative wounds. It is also shown in a large percentage of cases that the recurrence assumes the malignant type, even when careful examination has shown the original tumor to have been apparently benign. No definite theory has been advanced in regard to this phenomenon. I might suggest that more thorough examinations be made of serial sections of the primary growth, and by so doing there might be some portion of the specimen which would appear to have some malignant tendencies. The fact that, as a rule, the recurrence is not in the site of the original growth causes, at least, a theory upon the subject, in that the undue trauma of the vesical mucosa and submucosa, particularly so in cystotomies, sets into activity cells which otherwise might not be stimulated into proliferation and the formation of a new-growth.

The symptomatology and diagnosis are so classic that I will not get into a prolonged discussion. I would say in every case of painful and frequent micturition a cystoscopic examination should be recommended, as early diagnosis of tumor here, as elsewhere in the body, is essential.

Hematuria is recognized as the cardinal symptom of vesical papillomata; this is true, but it proves that the growth has advanced to a marked degree. I am of the opinion that, if the diagnosis be made early, before the presence of marked hematuria, there would be fewer cases of recurrence.

It is the consensus of opinion that a radical operative procedure is the only method of treatment. The methods most commonly employed are cystotomy by the suprapubic route, extraperitoneally or transperitoneally, operative treatment intravesically,

as first outlined by Nitze and advanced by others, and last, but not least, the fulguration method as first applied to papillomata by Beers and advanced by Keyes and others.

The case which will be presented through the kindness of Dr. Murphy is one in which the history is so characteristic that I will report it in detail:

Male patient, thirty-eight years of age. Family history negative. Previous history negative, patient denying all forms of venereal infection.

The symptom of his present trouble began more than seven years ago, when the patient noticed some disturbance of micturition, demonstrated as a pollakiuria with dysuria. Little attention was paid to this trouble until, about seven years ago, there was a sudden attack of hematuria which lasted over a period of two days and stopped as suddenly as it began. Little attention was paid to this. There was no examination, and consequently no diagnosis at this time. The patient had no more hematuria for about six months, when just as suddenly as the first attack the second began, and like the first there was no pain accompanying the bleeding. This stopped as suddenly as the first. Still there was no examination and no diagnosis. For several years these attacks came more frequently, lasting for a longer period and became more profuse. Two years ago last October the patient consulted Dr. Murphy. He went through a rigid examination, and following a careful cystoscopic examination a diagnosis of papilloma of the bladder was made. The growth was quite extensive; a large part of the bladder-wall seemed to be covered, yet it was pedunculated and arising from a single pedicle. The growth was situated on the posterior wall, immediately to the right of the left ureteric orifice, and, fortunately, not involving the orifice. An open operation was decided upon. Dr. Murphy performed a suprapubic cystotomy extraperitoneally and removed the entire growth by excising the portion of mucosa immediately surrounding the base. The base was cauterized, and the bladder closed with drainage. The patient made an uninterrupted recovery, with the exception of a sudden acute dilatation of the stomach—a post-operative gastric

tetany—which occurred the second day after the operation, and he was in a very serious condition for a day or two. He left the hospital perfectly well. Pathologist reported the specimen pure papilloma, non-malignant.

Patient gained weight and remained free from trouble for over two years, when he again noticed a small amount of blood in the urine. He paid no attention to this until he noticed more blood, and that the hematuria was persistent.

I was consulted. A cystoscopic examination was made. The entire posterior wall of the bladder was perfectly normal. To the left side, at the seat of the original papilloma, there was a perfectly normal scar, with absolutely no evidence of recurrence. From where was the blood coming? Examination of the anterior bladder-wall cystoscopically disclosed the trouble. At the site of the scar the result of the original cystotomy there was a distinct papillomatous mass hanging down into the bladder, and waving like seaweed in the ocean.

There was evident a distinct ulceration of a portion of the growth from which part the bleeding was taking place.

The cystoscope demonstrated two very important factors: first, that the recurrence of the growth was not at its original site, but situated well away from it, in the anterior wall of the bladder; secondly, demonstrated that the hematuria was produced from a distinct ulceration of the growth.

I give the history of this case because it is a typical one.

There is one thing I want to impress upon you more than any other, and that is where you have a hematuria *without pain*, intermittent in character and extending over quite a period of time, consider the case tentatively as one of papilloma, until from your examination you have reached a positive diagnosis of some other condition. Another feature of importance in papilloma of the bladder is early diagnosis, and I wish to take this opportunity to emphasize most strongly the importance of careful examination of all your cases suffering with any disturbances of micturition.

An early diagnosis in vesical papilloma is important, for upon this fact depend your form of treatment and your prognosis.



Fig. 135.—Shows position of patient upon a properly made cystoscopic table, with Bierhoff knee crutches. Patient's hip elevated. To the left, under the table, is a Wappler high-frequency apparatus, with connections for fulguration. This shows also the first step of passing the cystoscope into the urethra.



Fig. 136.—Shows second step of passing the cystoscope. The outer end of the instrument is brought well down between the patient's thighs to allow the short beak to take the natural curve of the urethra.



Fig. 137.—Shows method of irrigating the bladder. This is particularly important, especially when there is a marked hematuria accompanying papilloma. The thumb is held over the outer end of the cystoscope after the obturator has been removed. The fluid is allowed to run, filling the bladder.



Fig. 138.—Shows the thumb removed from the end of the cystoscope, allowing the fluid to escape. The thumb is then placed over the end, allowing the bladder to refill. This is allowed to escape again. This procedure is done until the fluid returns clear.



Fig. 139.—Shows the telescope carrying the fulguration wire in place and the operator locating the tumor and pushing the end of the wire into it. The spark is controlled by a foot-switch. Irrigation may be continued by opening the stop-cock to the right, if the medium in the bladder becomes cloudy. The entire intra-vesical operation is done directly under the eye.

If the diagnosis is late, then the chances of removing the growth by intravesical operation is small, and the surgeon must resort to a suprapubic cystotomy, which, from my experience, increases the possibility of recurrence.

In the majority of cases that I have operated by the open method there has been a recurrence. I have had eleven cases under observation. Of these eleven cases, eight are dead from malignant recurrence and metastasis, the deaths all occurring within eighteen months of the original operation. One apparently well with no recurrence after two years. One still alive, but with malignant recurrence, and one only a few months old and still free from recurrence.

In all these cases the microscopic findings were *reported* as benign papilloma, which was probably an error, as evidently the bases of the tumors—the point at which the malignancy most commonly shows—were not sufficiently carefully sectioned and examined.

With this record I have accepted intravesical operating as the safer method, particularly the method of fulguration by the use of the high-frequency current. The results following this mode of treatment have been almost perfect, but to obtain the best result an early diagnosis is paramount before the growth has become extensive, so that it can be attacked only by the open operation and probably has not undergone malignant change.

In 31 cases treated by the high-frequency current through the cystoscope I have had only one recurrence, and that was in a patient who refused to follow directions. He did not report for treatments regularly, he was a high liver, and indulged in a considerable amount of alcohol. In this case the recurrence was not malignant and fulguration has entirely cleared up the condition. The case is still under observation, and I am unable to state what will be the end result.

The technic of this work is comparatively simple—any one who is familiar with the use of the cystoscope can apply this form of treatment to vesical papillomata. One must, however, pay just as much attention to surgical principles as for a major operation. Special care must be exerted from the standpoint of

asepsis and antisepsis. The best cystoscope for this work is the Bransford Lewis universal instrument, for with its use one can accomplish more than with any other single instrument. With this cystoscope you can obtain either the direct or indirect approach as the case requires, through the passage of a single instrument, thus reducing to a minimum the dangers of trauma and infection.

The patient is placed in the extreme lithotomy position, as shown in Fig. 135. The external genitalia are thoroughly prepared by washing them with soap and water, followed with a solution of 1 : 500 mercury oxycyanid. The urethra is anesthetized with tablets of alypin, which are placed in the deep urethra through the Lewis tablet depositor.

The cystoscopes cannot be sterilized by boiling, as heat will soften the cement around the lens, causing them to leak, thus rendering the instrument unfit for use. Therefore the instrument is best kept in formaldehyd gas when not in use, then placed in a solution of 1 : 500 mercury oxycyanid for about five minutes. This mercury solution will not affect metal instruments, nor will it irritate the mucosa of the urethra or bladder. An irrigating tank filled with a solution of mercury oxycyanid solution 1 : 5000 is at hand to be ready for bladder lavage. The lamp on the instrument should be tested prior to the passage of the instrument into the bladder, to be sure that it is in good working order.

The operator prepares himself as for a major surgical operation. The cystoscope is lubricated with glycerin or any soluble lubricant, and passed along the urethra much like an ordinary sound, but to allow the short beak of the instrument to pass readily into the bladder the outer end of the cystoscope must be lowered well between the patient's thighs. Fig. 136 demonstrates this method.

The obturator is removed, and the bladder thoroughly washed through the cystoscope, as shown by Fig. 137.

The fulguration wire should be ready and in place in the telescope of the instrument. The fulguration outfit can be conveniently placed on a table near by and connected with the street current; a foot switch on the floor will regulate the spark. The

telescope is passed, and the wire passed onward into the bladder. The connection is made, and the operator is ready to begin his work.

The bladder should be filled with the irrigating fluid, and sufficient lavage must be done to render the fluid in the bladder clear, so that a definite picture can be seen. The tumor is located, and the wire gently but firmly passed into the base of the tumor, care being exerted to prevent the wire from piercing the bladder-wall. The spark is then turned on by means of the foot-switch, and the effect watched closely through the cystoscope.



Fig. 140.—Diagrammatic of the position of the cystoscope in relation to the papilloma. Showing also the manner of pushing the fulguration electrode into the base of the growth.

The entire base of the tumor can thus be treated at one sitting, as a rule. If the fluid in bladder becomes cloudy from bleeding, then the work may be accomplished with continuous irrigations, the irrigating tube attached to the cystoscope by means of a rubber connection, and then turned on, the fluid running into the bladder and running out continually. Fig. 140 demonstrates the method of applying the fulgurating wire to the tumor.

The high-frequency spark seems to have special action upon these growths. Usually after the first sitting the growth sloughs off and is cast off in the urine; this takes place for two or three days, and leaves behind a clear, healthy ulcer. At the second

sitting there should show a healthy condition of healing. The patient makes a rapid and perfect recovery, and in a short time a cystoscopic examination will show nothing but a scar remaining. Frequent cystoscopic examinations should be made for the purpose of ascertaining the presence of recurrence. I feel that the patient can be discharged if there is no recurrence after one year.

From my observations of results following the two methods of treatment I am forced to admit that intravesical operating is the method of choice in all cases. Its advantages over the open operation are many: the recurrences are less than in the open operation,—there is no operative mortality,—the patient can attend to his daily vocation, as he is not confined to the hospital, as he would be for the open operation.

In closing, gentlemen, I wish to be clearly understood in relation to the importance of early diagnosis, and I wish to insist upon the fact that in every case of a disturbance of micturition that the patient be rigidly examined and every method employed to elicit an accurate diagnosis. Not only is this important in cases of vesical papilloma, but in all classes of cases with symptoms pointing to the genito-urinary system; for with a correct and early diagnosis the end results will be better, mortality will be decreased, and the advantages of surgery placed upon a higher level.

I wish to thank Dr. Murphy for the privilege of presenting this case to you.

[Since demonstrating this case in the clinic I wish to state that he has made a complete recovery. Owing to the unusual position of the tumor, it was exceedingly difficult to reach it with the fulguration wire, and required about eight treatments before I had the growth entirely removed. The bleeding completely stopped after the first application. The patient's bladder at present is normal. He did not miss a single day from his work, and at present his general condition is good. A tabulated report of my cases of vesical papilloma will appear elsewhere later.—L. W. B.]

AMPUTATION NEUROMA WITH ASCENDING NEURITIS; DIVISION OF RIGHT HALF OF CAUDA

REMARKS ON NEUROLOGIC PHASES BY DR. CHAS. L.
MIX

HISTORY

C. M. Patient, male, aged fifty-one, entered the hospital November 2, 1913, because of severe pain in the stump of his right leg, which had been amputated through the upper third of the thigh nine years ago. His clinical history is as follows:

At nine years of age, while running, he sprained his right knee. This caused him severe pain at the time. He continued to use the limb, although he had pain in the knee occasionally, but never severe enough to incapacitate him. At twenty-five years of age (1887) he began to have severe pain in the knee, exaggerated on walking. He then began to use a cane. He got along fairly well, but the knee kept gradually getting weaker and more painful until ten years ago (1903), when he had to use a crutch and keep the foot from touching the ground. He could not put any weight on the leg. At this time he was told that he had tuberculosis of the knee and an amputation was advised.

Nine years ago (1904) the leg was amputated in the upper third of the thigh. He does not know how the nerve was treated at the time. The wound healed by first intention in a few weeks. Immediately after recovering from the effect of the anesthetic he began to have severe pains in the right limb, seemingly beginning in the toes and extending upward. He also had occasional pains along the posterior surface of the stump. He began to use bromids to get relief from the pain. This pain continued for six months, at which time his doctor told him he had a neuroma

on the sciatic nerve. An operation was performed, and about four inches of the lower end of the stump of the sciatic nerve were removed. The wound healed in three or four weeks.

Immediately after this operation he began to have a recurrence of the former symptoms, and in addition he had spasmodic pains, which he referred to the big toe of the amputated foot. He says it felt as though some one was sticking a knife through it and then removing the knife. These attacks lasted for from twelve to twenty-four hours, but he had almost constant dull pain in the stump. After three years (1908) his doctor told him that it was impossible to relieve him of his pain. He was again operated on, however, under local anesthesia, and another piece of the sciatic nerve was removed. After this operation the pains were just as severe as before, and continued so for the next year. In 1909 he was again operated on. The nerve was drawn down, divided, and allowed to retract into the stump. This did not relieve the trouble, however.

The last operation was done three years ago (1910). At this time an incision was made along the right side of the spinal column, at the lumbosacral joint, and the posterior spinal nerve-roots were said to be divided, but he was not relieved of his pain. During the last year he has been taking codein constantly. When the attacks of pain are mild, he takes two grains, and when the attacks are severe, he takes three grains, repeating the dose if necessary. He has taken as much as 12 grains in the course of twelve hours without getting relief from the pain.

Previous history: He had typhoid fever twelve years ago; lasted eight weeks; no complications. Has stomach trouble off and on. Has slight hernia and hemorrhoids at present.

Family history: Two aunts died of tuberculosis of the lung. No history of carcinoma.

COMMENTS AND OPERATION

DR. MURPHY (November 9, 1913): You have heard the history without the details of the structures involved at the present time. Dr. Mix has gone over this case, and I have asked him to give you the details of the neurologic phases of the case.

DR. MIX: The history of the case as given by the intern is quite complete, with the exception of the anatomic details, the result of the examination. A most noteworthy point in the history is that immediately after he recovered from the anesthetic, after his leg had been amputated, he felt pain in the ball of his foot, and shortly thereafter in the big toe and in the ankle-joint. This pain did *not* come on several weeks after the amputation; in other words, the pain appeared before there had been time for the development of a neuroma. A pain following so closely upon an operation indicates, therefore, not the ordinary hypertrophy of the severed ends of the nerves, but rather an irritation of the ends of the severed nerves. The pain the very day following the operation was located particularly along the inner side of the foot, and in the ball of the foot, in the territory of supply of the external plantar nerve, which comes from the posterior tibial. There was also an involvement of the inner side of the great toe. This territory is supplied by the internal saphenous branch of the anterior crural nerve. Furthermore, there was a great deal of pain between the great toe and the second toe, in the territory supplied by the anterior tibial nerve, which is a branch of the great sciatic. Some of the territory of supply of the musculocutaneous nerve in the instep was also painful. The musculocutaneous, you remember, is a branch of the external popliteal coming from the great sciatic. We, therefore, have in this man's case signs of disturbance not merely in the territory of supply of the great sciatic nerve, but also irritation in the territory of supply of the anterior crural nerve, which comes from the second and third lumbar segments of the cord.

The conclusion which was originally made by the surgeon who did the first, second, and third operations, was that a neuroma had formed in the end of the great sciatic nerve. It was on that account that a second operation was performed, and from two to four inches of the lower end of the sciatic nerve were removed. Of course, this operation was doomed to failure from the very beginning from the fact that much of his pain was in the territory supplied by the internal saphenous branch of the an-

terior crural nerve, which comes from a much higher level, and which the surgeon either overlooked or ignored.

The pain having again reappeared, the next step taken was to remove another piece of the sciatic nerve, the same error being again committed. No benefit having followed this operation, an incision was finally made along the right side of the spinal column, and we are informed the origin of the sciatic nerve was cut. According to the history of the case given to us by the patient, the posterior roots of the sciatic were severed.

Examination of the patient's sensation in the thigh shows that the territory of supply of the ilio-inguinal and iliohypogastric nerves from the first lumbar segment of the cord is intact. The supply of the external cutaneous nerve from the second lumbar segment is also sensitive. The territory supplied by the internal cutaneous and the middle cutaneous branches of the anterior crural nerves, in that little which is left of the thigh, is sensitive. I learned from the patient, independently of the intern's history, that at the last operation there was a resection of the third, second, and first sacral nerves, and presumably some of the lower lumbar nerves, but it is quite evident, from the examination of the patient and the preservation of his tactile sensation, that either the resection of the lumbar filaments was not complete or that some of the filaments which were thought to be lumbar filaments were not in the severed portions. Inasmuch as the disturbance involves the inner margin of the big toe, the supply of which is from the internal saphenous, it is evident that if this man is to be helped, the roots must be divided as high as the second lumbar.

What is the origin of this man's trouble? Of course, it is not an ordinary neuroma, such as occurs in an amputation stump. We must assume either that two such neuromata exist, one on the anterior crural and the other on the great sciatic, or that none exists and that another etiology is present. In thinking this case over, it has seemed to me likely that there took place at the time of his original amputation an ascending neuritis. This ascending neuritis seems to have involved the whole of the sacral and lumbar plexuses on the side of the cord of the amputation.

There is no evidence that the cord itself is involved in any way. The conductivity of the opposite leg from the point of view of the nervous system is perfect. The conductivity of the amputated stump is, apart from the irritation and sensory loss due to the last operation, otherwise normal. The great difficulty seems to be the intolerable neuralgia. In view, therefore, of the fact that the pain almost immediately followed the recovery from the anesthetic, that it has been continuous and in no wise helped by such operations as have been done, we are of the opinion that the trouble is due to an ascending neuritis. It is on this account that we have concluded that a sort of exploratory laminectomy be done; that the nerve-trunks in question be exposed, and that if they be found, as we think them to be, diseased, they be severed.

I might say in conclusion that the patient came to us with a letter making the suggestion that perhaps the pain was "cortical," and not due to any disturbance in the amputated thigh. I think if you will review the history of the localization of the painful areas in his case and the constancy of these painful areas, that it will be very evident that the malady cannot be "cortical" in origin. When symptoms follow anatomic lines as clearly as these symptoms follow them, they are clearly and directly the result of disturbances of the peripheral nerves. It is impossible, therefore, for the symptoms to be cortical in origin. They must be due to an involvement of the sacral and lumbar plexuses.

DR. MURPHY: Dr. Mix has made the matter quite clear. If you will mentally unfold the caudal zone, you will find that it will aid in keeping your anatomic relations fairly clear in estimating the nature and extent of injuries below the cauda, because that is what you have to deal with. What do I mean by unfolding? Take your anterior crural nerve, the largest branch of the lumbar plexus: it supplies the iliacus, pectineus, and all the muscles on the front of the thigh except the tensor vaginae femoris; sends cutaneous filaments to the front and inner side of the thigh and to the leg and foot, and articular branches to the hip and knee. It arises from the second, third, and fourth lumbar nerves. The great sciatic nerve arises from the fourth and

fifth lumbar, and the first, second, and third sacral nerves, supplying nearly all the integument of the leg, the muscles of the back of the thigh, and those of the leg and foot. It divides into the internal and external popliteal nerves. Simplifying it in that way you have a fairly good estimate of where your relationship should be, what nerves should be involved, and how you can best handle the condition.

This man has his pain in two places. The last time I asked him, "Where is most of your pain?" he said, "Most of the pain is in the neighborhood of the ball of the foot," that is, as we interpret it, along the distribution of the internal cutaneous or long saphenous nerve, a branch of the anterior crural, and the internal plantar, a branch of the internal popliteal. But he says, "In the joint of the ankle I also have pain." If there is any pain in the ankle, the musculocutaneous nerve is involved, because that is where the nerves of the ankle-joint come from, and the internal popliteal is given off from the sciatic. The front of the ankle is supplied by the anterior tibial and musculocutaneous, a branch of the external popliteal nerve, which also comes from the sciatic.

It is our intention to expose the cord and then decide on the further course we will pursue. The patient is lying on his left side, with a sand-bag under him, so as to arch his spine in the dorsolumbar zone. The arms are in front of the body, not under it. The skin has been sterilized with a 3.5 per cent. strength tincture of iodine, and the sheet and towels are in place, so that the field of operation alone is exposed.

We make a longitudinal incision over the spinous processes of the eleventh and twelfth dorsal and the first and second lumbar vertebræ, cutting down onto the processes. The soft tissues are retracted to either side; the muscles are separated from the bone with a periostome, so that the spinous processes are stripped clean and in plain view. With a large rectangular bone-cutting forceps the four spinous processes mentioned are removed close to the laminae, and then the corresponding laminae are excised with the rongeur forceps. All the bleeding is easily checked by compression with small gauze sponges.

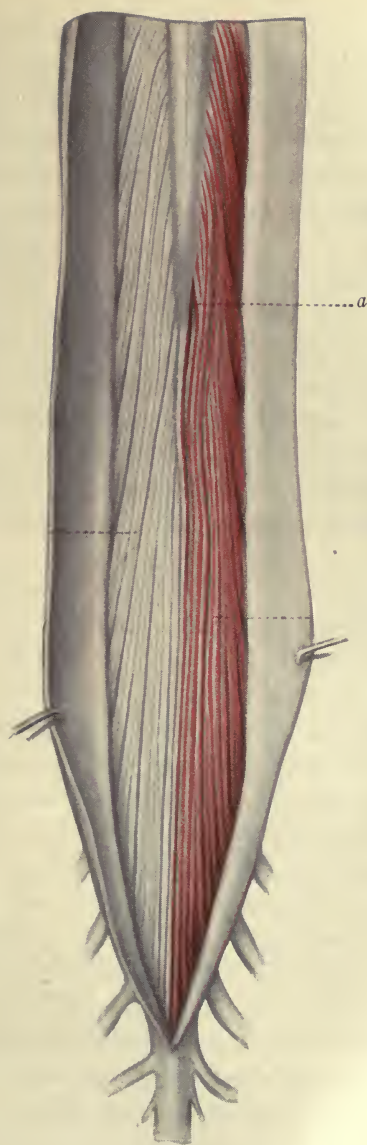


Fig. 141.—The bright color is almost an exact reproduction of the color in the half of the cord on the affected side. The light color represents the appearance of the color on the opposite side. Shows pain was due to ascending neuritis.

We are below the conus. There is the fibrous layer outside of the cord lining the canal; that is a good exposure. There is a little oozing from the bone. By pressing in a Horsley sponge, that is easily stopped. That is just a venous oozing and has scarcely a plus pressure; now I have perfect quietude so far as hemorrhage is concerned. We want this space perfectly free from bleeding when we open the cord.

I divide the dura lengthwise, and as I do so the cerebrospinal fluid escapes freely. The pressure will soon come off. The lesion is on the right side of the conus. See the difference in the cord! The right side is congested, tense, and inflamed, and the left side is soft and pliable and of normal color. That shows up well and explains why this man has had continued pain. He has an ascending neuritis, taking its origin in the stump of his sciatic nerve and extending up into the conus, involving the right side only.

Now the question is whether we had better cut the right half of the conus across and paralyze the stump and half of his external anal sphincter, which is supplied by a branch from the anterior division of the fourth sacral and the inferior hemorrhoidal branch of the internal pudic. That will paralyze motion and sensation in the stump. The nerves can regenerate for motion if the stumps are properly approximated, but never for sensation. It will put all that side out of commission. I am retaining that inner fasciculus because that is the one I think folds in and comes around to the internal sphincter. You see I am leaving that one in the middle that goes down into the internal portion. We pass a nerve hook under the inflamed right half of the cauda and raise it up (point *a*, Fig. 141). You note what a beautiful bright pink color it is compared with the left half, white and glistening. Then we divide it transversely beyond the zone of inflammation. That should effectually check the neuritis of the sensory axons from traveling higher up and free this man from his pain.

That was certainly a beautiful picture. That is the first time I ever saw that condition, and it shows, as stated in the discussion before the operation, that the pain is due to the ascending neuritis and not to a neuroma. (See Fig. 141.) It was

so striking that you could read it from afar. This looked like an obscure proposition, but we have gone at it in a careful way, and you saw how easily it was solved.

Now I will close the opening in the dura, approximating the edges accurately with fine plain catgut. The remainder of the wound is closed with silkworm-gut sutures and horsehair for the skin incision. Although we have taken out the spinous processes, this man's back will be just as strong as it ever was. I have never seen the slightest inconvenience result from the removal of the spinous processes and laminae. A plain dressing is applied, held in place with a binder.

Let the record show that all the fibers that were congested were in the right half of the cauda; they were divided, and half an inch of the portion was removed for microscopic examination.

Let the record show further that the fibers' fasciculi, filaments making up this half of the cauda, were congested and fiery red, in most pronounced contrast to the normal fibers on the opposite side; that these filaments could easily be picked up separately, but that it was decided to remove all the external filaments and a part of the central filaments in order that the pain might be checked; that before the operation it was thought that we could keep the center fibers of the cauda as they come down from the conus, and by keeping these center fibers we would have the portion that goes to the anus retained, but on account of the inflammatory condition it was decided that we divide all the inflammatory portion of the cauda; that it is barely possible that some of the white fibers may still be a part of the center fibers in the right half of the conus, and, if so, will supply the anal zone; that it was hoped we would be able to conserve the anal zone supply, but in any event he will have control of half of the external sphincter, which is often ample.

Let the record further show that Sister reports the Horsley sponges counted and correct.

What will be the hope for the distal supply that was cut off? Will there be a regeneration of these divided nerve-axons? Let us analyze that situation. When the motor nerve neuron leaves the spinal cord, it receives what? A neurilemma. Every nerve

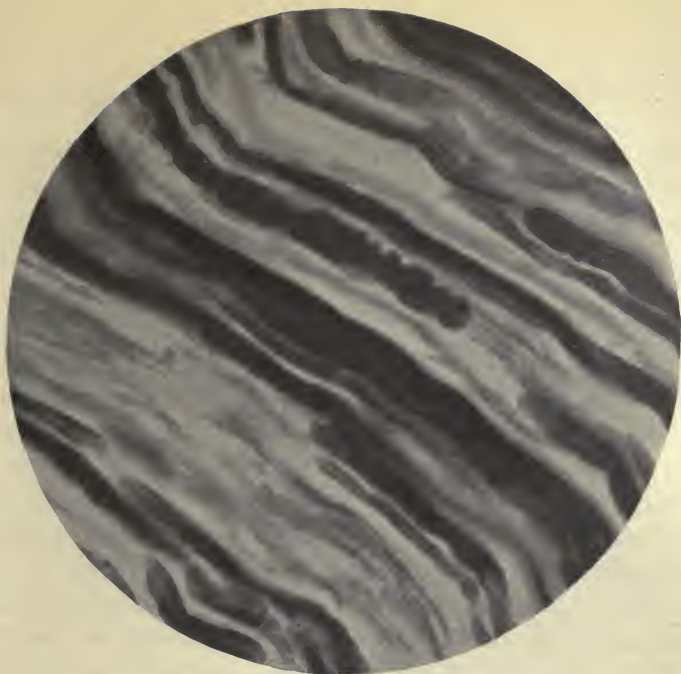


Fig. 142.—Weigert-Pal stain. Shows marked atrophy of nerve-fibers, broken-up disintegrated medullary sheaths, with irregular shaped masses of myelin.

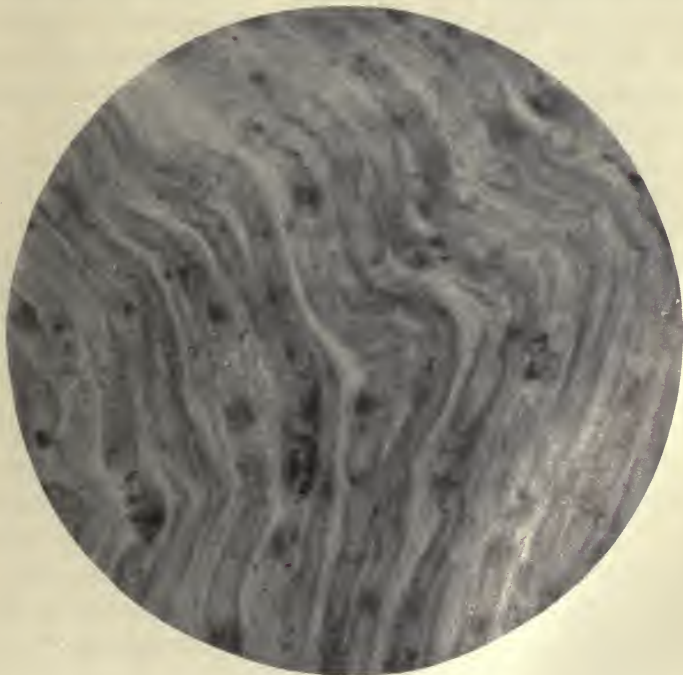


Fig. 143.—Marchi stain. Shows degeneration of nerve-fibers; nearly all the fibers show fat-droplets in their myelin sheaths.

that goes out from the spinal cord receives a neurilemma investment—that is proposition No. 1. Proposition No. 2 is, where are the central cell-bodies of all the motor axons that are represented in these fibers? The motor neuron central cell-bodies are situated in the anterior cornua of gray matter in the cord; so if you divide a motor nerve outside of the cord, as it has a neurilemma, all you need to do is to reunite it and regeneration takes place and function returns. That is all with the motor axon, but these motor axons have no relation to the patient's pain.

See the difference when you come to the sensory axons here! The sensory nutrient cell-body is situated in the anterior root ganglion inside the spinal foramen. In the sensory neuron you have a centrifugal portion of the cell axon coming down as an axon of sensation in the mixed nerve-trunks, and you have the proximal axon portion going into the cord and up in the columns of Goll and Burdach. Both the centrifugal and centripetal axons have a neurilemma when they go out of the ganglion, but when the central axon extends up in the columns of Goll and Burdach, it has no neurilemma, so if you cut the centripetal axon off between the posterior root ganglion and the cord it degenerates centripetally and you never have a regeneration of the fibers in the cord. Remember that a divided nerve axon degenerates in a distal direction from the central cell body. That is all you have to remember. Now the central cell-body of a motor neuron is in the anterior cornua of gray matter. Divide the nerve outside the cord, and degeneration takes place in a distal direction, but regeneration will occur if the nerve has a neurilemma. All motor nerves have a neurilemma outside of the cord and outside of the brain; therefore they all regenerate. If you divide a sensory nerve outside of the ganglion, it degenerates in a distal direction, but it will regenerate if you approximate it; but if you divide it inside the ganglion, then it degenerates proximally into the cord, where it has no neurilemma and it will never regenerate.

All you have to remember is to ask yourself what nerve-roots have a neurilemma. All the cerebral nerves except those of special sense have a neurilemma. Therefore the nerves that will

not regenerate are the optic, the auditory, the gustatory, etc. Those are the nerves of special sense. Why do they not regenerate? Because they have their central cell-body in the organ of special sense and they have no neurilemma. Their proximal axons run back to the brain, so that when you cut the optic nerve it never regenerates. Ordinarily the cerebrospinal nerves, except those of special sense, always regenerate if you approximate them. You get a reestablishment of function uniformly if the divided ends are properly united aseptically. There is no excuse for not reuniting a divided nerve. You will be held just as responsible in the courts in the future for not reuniting a divided nerve as you were formerly held responsible for not putting the ends of a fractured bone in apposition, because they as uniformly reunite and have a reestablishment of function when they are approximated as do divided or fractured bones.

[Healing *per primam*. There was much immediate relief of pain. Sensation was absent all over the stump. In a few weeks he claimed there was a return of the same pain. This does not correspond with our present knowledge of the sensory nerve-root phenomena. The question is, has he the pain or has he the drug habit, which he now has returned to? I am in favor of giving him the benefit of the doubt, and accept a return of the pain, but I cannot explain it. Professor Zeit has furnished us the accompanying photomicrographs showing the process of ascending degeneration in the axons in the fascicule removed from the cauda.—ED.]

NEUROMA OF THE ULNAR NERVE, RESULT OF CICATRICIAL COMPRESSION FOLLOW- ING UNRECOGNIZED FRACTURE

HISTORY

PATIENT, Dr. E. F., aged forty-six years, entered the hospital January 28, 1914, with the following history:

Family history negative to tuberculosis. He believes a grandmother died of carcinoma of the stomach. Past history negative.

In the fall of 1902 the patient noticed that the adductor muscle of the left thumb was weak and atrophied, and that he was unable to abduct the index-finger. During the past three years the interossei have been atrophying, more so in the past six or eight months. Since the summer of 1913 the atrophy appears to have progressed more rapidly. The hypothenar eminence becomes numb if any object is held between the thumb and fore-finger for a long period of time. The lumbricales are atrophied. The patient states that to him it appears as though his grip has become weaker in the left hand during the past three years. He cannot remember anything regarding a former fracture of the arm or elbow.

COMMENTS AND OPERATION

DR. MURPHY (January 29, 1914): This is a very interesting case, of a class of which we have had two within the last two weeks, and they are as much alike as two peas. We have not seen a case just like these in years. He has an atrophy of all the muscles supplied by the ulnar nerve, but more particularly the lower ones—the hand muscles—meaning what? That something is interfering with the function of his ulnar nerve.

The most common condition interfering with the function of

the ulnar nerve in the hand is a seventh cervical rib. That not only interferes with the nerve function by a pressure neuritis, but also with the arterial circulation, not with the venous circulation. They never have an edema or swelling of the arm or hand. The condition of the muscles of the hand as described by the doctor is typical of a seventh cervical rib compression. The rib compresses the artery against the scalenus muscle, but it never presses the vein. Why? Because the vein is in front of the scalenus anticus muscle, and as the rib presses forward it must compress the artery against this muscle, but the vein can escape. One of the early symptoms is numbness of the hand, particularly when it is washed in cold water; it becomes white and remains white for many minutes, even to an hour, when the color returns. In the later stages of the disease the fingers may become gangrenous from ischemia combined with trophic nerve disturbances. That is not the condition present here. This man has no seventh cervical rib.

When you look at this elbow with the arm extended, you will note that there is a prominence of the internal condyle. It is anterior to where it should be. To the inner side of that point is a bony tubercle. On that has developed an enlargement of the nerve,—a thickening,—just exactly as we saw it in the other case.

The arm has been prepared in the usual manner, and a constrictor will be applied. Then, with the arm flexed, we make a longitudinal incision on the inner posterior aspect of the arm, so as to get a good exposure of the ulnar nerve. We retract the wound edges and there is the nerve shining through. Is not that pretty? (See Fig. 144.) Note the enlargement as it lies in the groove. There is a little mass of bone just to the inner side, which is causing the enlargement of the nerve by continuous irritation. It is blocking the nerve up quite a distance. This neuroma is about two inches in length and of about the thickness of a lead-pencil. The nerve comes back again to its normal size about one inch below the condyle.

What are we going to do? There is just one thing to do—not to take the neuroma out, because it would destroy the function of the nerve, but remove the nerve from where it is being traumat-

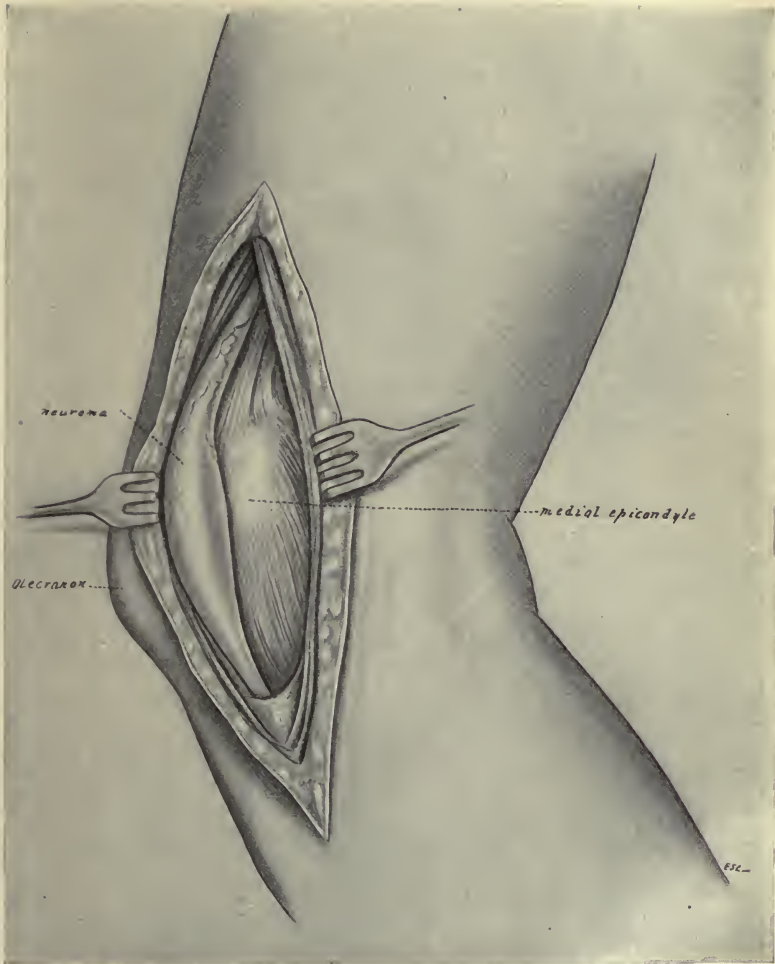


Fig. 144.—Neuroma of ulnar nerve in groove between olecranon and internal condyle—the result of irritations due to exostoses. The blood-vessels at the upper end of the neuroma are dilated.



Fig. 145.—Neuroma of ulnar nerve. The patient did not recall injury to his elbow, but the skiagram shows that a fracture was sustained. The tip of the internal condyle is broken off and has not united with the shaft. The external condyle appears to be absent. The shaft of the humerus above the condyle is greatly thickened. The radius and ulna are in normal relationship to the humerus.



Fig. 146.—Neuroma of ulnar nerve. Lateral view. There is a considerable enlargement of the coronoid process of the ulna, in fact of the upper inch and a half of both the radius and ulna. The lower end of the humerus appears to be normal.

ized to a position of safety. We will throw it clear out of its position in this groove to a free location just in front of the condyle. That is the way I handle all these cases. We will bring this nerve right out into the loose connective tissue, so that it cannot be further traumatized. That is all—just put it out of harm's way. It will take about one year and a half for the function to return in this nerve; by that time absorption of the inflammatory exudate around the axons will have disappeared.

The faradic current, which we are now applying below the neuroma, shows that the axons have not been divided nor are they degenerated, because the muscles of the hand supplied by the ulnar nerve contract to this current. There was an obstruction to transmission of voluntary impulses, but the axons were not divided or degenerated, therefore function will return. The swelling on that nerve is just as sensitive to pressure as is the eye when you touch it with the finger.

To what is that enlargement due? To an excessive production of connective tissue of the neuroglia type around the axons. That connective tissue first increases and produces a neuroma; then gradually contracts and stops the conductivity of the axis-cylinders, but does not cause degeneration. Stop the irritation, and absorption finally commences to take place, and the result is a restoration of function. These axons are not degenerated. The changes that have taken place here are trophic changes, showing that the trophic axons are the first to be affected by an obstruction. The trophic axons atrophy long before the motor or the sensory axons in this class of case, and it is the same when it is a seventh cervical rib. Motion will continue long after the muscles atrophy. Patients with a seventh cervical rib will tell you that they can pick up objects and hold them for a short time only, then they must drop them. The lesions cause a suspension of function.

We displace this nerve into the soft tissues and throw a fat and fascia flap over the ulnar groove, so as to prevent the nerve from becoming fixed to the fine connective tissue of the joint capsule or periosteum, tacking the flap in place with catgut. The skin wound is closed with horsehair and the usual dressing is

applied. The bony mass which caused the irritation of this ulnar nerve is an exostosis (point *a*, Fig. 145), either from a previous fracture, not recalled by the patient, or from a failure of union between the embryonal center of ossification of the condyle and the shaft, as shown in Fig. 145. The radius is luxated slightly forward in the lower sigmoid cavity. (See Fig. 146.)

NEUROMA OF ULNAR NERVE, THE RESULT OF TRAUMA INCIDENT TO FRACTURE AT ELBOW

HISTORY

PATIENT, Mr. R., aged thirty years, entered the hospital January 6, 1914, with the following history:

Family history negative to tuberculosis and carcinoma.

Past history: Had children's diseases. When the patient was four years old (1888) he fell, striking on his left elbow. The arm was useless at once. A doctor was called and dressed the arm in a splint; the patient does not know how long the splint remained on. When the splint was left off, the arm was stiff at the elbow, but with forced motion and exercise movement was reëstablished. The arm then was apparently restored to usefulness except for the present condition. He has had pain in the arm from the time of the injury when he was four years old. Six months ago the pain commenced to decrease in severity.

At that time the patient noticed that the third and fourth fingers, on attempted flexion, were in a more extended position than the rest of the fingers. These two fingers frequently became numb, the numbness extending as high as the styloid process of the ulna. He cannot bring the fingers of the left hand together in apposition while in extension, nor straighten them without a hyperextension of the third and fourth. There are more or less absence of adduction of the thumb and atrophy of the hypothenar muscles; in fact, all the muscles of the hand are atrophied. The grip in the left hand is less than normal.

COMMENTS AND OPERATION

DR. MURPHY (January 8, 1914): When the hand is straight, he cannot bring the fingers together. What does that mean?

That the lumbricales and interossei are atrophied in a superlative degree, in fact, all the short muscles of the hand except the extensor and abductors of the thumb and the first two lumbricales are atrophied. The adductor of the thumb, lumbricales, and the hypothenar eminence are all atrophied. The history states that the hand grip is weakened. That is not true. He can grip with only part of his hand. The thumb is all right, but he cannot bring his thumb to do what? He has grip in the little and ring fingers. That shows that his adductor muscle is free, and that the lumbrical muscle which pulls the thumb over is free. He can pull half as strong on the one and only a little on the other. That means what? That he has an atrophy of all the small muscles of the hand. Remember all the small muscles of the hand, with the exceptions mentioned above, are supplied by the ulnar nerve. Therefore this man has half a grip in this hand. That tells the story that it is a lesion of the ulnar nerve.

He had a fracture of his elbow when he was four years old, and he still has a non-union of the external condyle, with a backward and inward luxation of the internal condyle. You may remember my saying that these cases rarely occur. Just when you make a law you find an exception to prove the rule. His external condyle is entirely loose, and he has a posterior luxation of his internal condyle. He also has a traumatic neuritis of his ulnar nerve, due to an osteoma under the ulnar nerve resulting from this fracture, and showing itself now, twenty-six years after the fracture occurred.

There is one other condition, remember, that could cause practically the same symptoms, and that is a cervical rib. The skiagram of this patient shows that he has no cervical rib. We made that skiagram purely for the purpose of exclusion. He has a thickening of the ulnar nerve over the internal condyle. It is also very sensitive to pressure.

We will expose the ulnar nerve by a longitudinal incision to the inner side of the condyle. Because of the great luxation of the condyles there is difficulty in outlining his ulnar nerve to show just where it is compressed. Can you see the thickness of that bone? That is due to the luxation backward of the internal



Fig. 147.—Neuroma of ulnar nerve. Fracture of external condyle of humerus with non-union, and deformity of internal condyle with excessive bone formation, causing the constant irritation of the ulnar nerve, which led to the formation of the neuroma.

condyle. That is what gives the pressure on the nerve. It is just too long a distance around this condyle for the nerve to escape repeated impressions in flexion of the arm. We must luxate the ulnar nerve up in position, just where it comes across the internal condyle. I am sure that the faradic current will show that he has only a suspension of transmission due to compression of the axons, not a degeneration of the axons.

You may recall the case we had recently where there was a transverse scar across the spinal cord, compressing it sufficiently to suspend transmission of impulses. When we applied the faradic current to the cord below the scar, all the muscles of the leg responded, notwithstanding a paralysis of five years' standing. When the current was applied above the scar, none of the leg muscles responded. When we divided the scar tissue, freeing the cord of pressure above the line of scar tissue, the leg muscles responded, showing that the axons were compressed but not divided, because if they had been divided, they would have been degenerated below and there would have been no transmission of impulses either below or from above after division of the scar. It was a suspension of transmission and not a degeneration.

As we retract the wound edges the ulnar nerve comes into view. It is three times its normal diameter at the point of irritation, in the groove to the inner side of the olecranon. Now we will see how many axons are living by testing the nerve with the faradic current above the neuroma. He has transmission through it, just as we told you before the operation, because he had feeble action in some muscles supplied by this nerve and not in others. The nerve was held in a fixed position over the edge of the displaced condyle. There is where it began to be traumatized.

We will free it from its bed and throw it around and up into this connective tissue, to the inner side of the internal condyle, so that it will not be traumatized again. The neuroma is about two inches in length and about three times the diameter of the normal nerve. Now that it is freed, it transmits impulses faradic, so that the atrophy of the affected hand muscles must

have been due to the connective-tissue compression of the axons in that nerve, particularly the trophic elements.

If we were to make sections of this nerve, we would find living axons and an excessive quantity of connective tissue which is slowly strangulating these axons and obstructing the transmission of impulses. By recalling your anatomy of the hand you get a splendid picture of this lesion. His lumbricales and interossei on the ulnar side and the long flexors of the little and ring fingers are supplied by the ulnar nerve, the remaining corresponding muscles by the median. The adductor of the thumb is also supplied by this nerve. That is the reason this man had only half a grip, if we may describe the condition present in those terms.

With his nerve protected from injury he will get a complete recovery of function by a fat and fascia flap placed beneath it. [Primary healing.—Ed.] The wound is closed with horsehair, and the usual dressing is applied. The arm will be carried in a sling.

INTERNAL HEMORRHOIDS WITH SEVERE BLEEDING AT STOOL, THE RESULT OF A SMALL SLIT IN A HEM- ORRHOIDAL VEIN

ANEMIAS: PRIMARY AND SECONDARY; DIFFERENTIAL
DIAGNOSIS BY DR. CHAS. L. MIX

DR. MURPHY (January 13, 1914): We have two cases in the house very much alike in facial and blood appearances, as well as clinical history. One I have been able to diagnose and the other I have not. The man I am going to operate on this morning has a leukopenia of 4800, with 1,728,000 reds; hemoglobin, 12 per cent.; and a blood-pressure of 121. He is a giant in stature, measuring six feet four and of splendid physique. He said that he began to have bleeding from his hemorrhoids about two months ago. On questioning him further he said it was two and one-half months, and before I got through examining him he stretched it out to four months, and the fact is that it was six or eight months since the severe bleeding began and for several years it has been intermittent. He does not bleed from his bowels except when at stool.

HISTORY

Patient, F. K., aged forty-eight, entered the hospital January 9, 1914, because of intense anemia and fainting spells. Off and on for the past twenty-five years the patient has been troubled with bleeding hemorrhoids. The attacks of bleeding have increased in number and severity in the past six or seven years. For the past twelve months he has bled at least once a day, but only while at stool. He has had no pain. The blood passed is usually bright red in color; at times it is dark colored, but never passed as clots. The patient feels weak after each bowel evacuation; he has had constipation and diarrhea alter-

nately; the latter would occur about once a month, and last for a few days. He has lost little in weight, but he has become very pale, weak, and has palpitation on the slightest exertion. His appetite is good.

In the summer of 1913 he had a bad taste in his mouth, was constipated, had a poor appetite, and his complexion was very sallow. One day, while at work, he suddenly became somewhat dizzy and faint and had a tachycardia. After a few moments he was able to continue. He consulted a doctor then, who gave him calomel and kept him in bed a few days. The attack passed off and the sallow color disappeared within a few days. He has had two similar attacks at different times since. He frequently took calomel. He has had no pain, no chills, no fever. He has not vomited, although he was nauseated for an hour or more after the onset of the attacks.

The last attack commenced a few days before Christmas, 1913. At that time the patient was constipated, had a poor appetite, and a bad taste in his mouth. On Christmas morning he gradually became faint; his heart-beat became rapid and very weak. He paused in his labor for five minutes, when the faintness disappeared. He felt very weak; his heart still beat rapidly. He did not go to bed, however, until January 2, 1914. He had occasional attacks of nausea from the third night on, and about the fifth night, when at stool, he vomited, without apparent reason. Three or four days later, when at stool, he again vomited. A few days later he vomited again, without cause. There was no blood in the vomitus. He never had any pain.

He has had to urinate twice during the night in the past year, but had no pain on urination and no blood in the urine. At times he urinated frequently for a few days, then returned to normal. Neither his ankles nor his legs swell. His complexion is a green lemon-yellow; his lips and conjunctivæ are white and bloodless.

PROCTOSCOPIC EXAMINATION.—January 11, 1914, with the eight-inch proctoscope, the mucosa of the rectum is seen to be white and anemic. There is a blood spot three inches up, but this is believed to have been carried there, as no ulceration exists

and there is no blood in the feces. There is an erosion in the hemorrhoidal region, but no bleeding vessel is seen.—J. B. M.

A careful examination shows no ulceration from which the blood might come. He never had tenesmus nor slimy stools.

Examination of the blood: Leukocytes, 4800; erythrocytes, 1,728,000; hemoglobin, 12 per cent.; blood-pressure, 121. Differential leukocyte count: Lymphocytes, 18 per cent.; large mononuclear and transitional, 4 per cent.; polymorphonuclear neutrophils, 76 per cent.; eosinophils, 1 per cent.; basophils, 1 per cent. Differential erythrocyte count: Poikilocytes, few; megalocytes, several (merely the rings—shadows); microcytes, several in field (denoting secondary anemia); normoblasts, 3 (three during differential white count); granular degeneration cells, many; polychromatophilia, many cells. This is the blood-picture of secondary anemia though he looks like a patient with pernicious anemia.

COMMENTS AND OPERATION

DR. MURPHY: Yesterday we had him pass a bowel movement for immediate inspection, and it was a formed stool, comparatively small, but it stood up in a pool of bright red blood, showing that it was a bleeding from the hemorrhoids. He has reached a superlatively anemic stage. He cannot walk without fainting, and has palpitation on the slightest exertion. We have a woman in the house in practically the same condition, although I have not seen her stool. I have never seen a more typical picture of this kind of case, both in the clinical history, physical and blood findings, and bowel movements—just a small formed stool in a pool of blood.

This is the third case of the kind I have seen. We had, some years ago, a lawyer who came to me from Dallas, Texas. When I examined him with the high proctoscope I could not find in the rectum a cause for the bleeding. His hemoglobin percentage was not so low,—about 60 per cent.,—but he had that peculiar green-yellow color that is so characteristic of a severe secondary anemia. I could not convince myself that it was a case of pernicious anemia, and examination of rectum revealed

no ulcer or bleeding point to account for the anemia, so I had him consult Dr. Sippy, who made a thorough examination of the patient, but failed to find where the blood was coming from. Finally he placed the man over a bed-pan and had him strain to cause an evacuation, and when his bowels moved, the blood shot out from the side of the rectum as from a syringe. There was just a little slit in the vein, which could not be seen on examination, as the edges folded into an entropion position when he ceased straining and turned out when he strained; this was what obscured the case on physical examination.

The history of that case was as follows: Male, aged thirty-four, entered the hospital October 18, 1908, because of hemorrhage from the rectum. Seven years ago he first noticed blood in his stool. He had on an average one bowel movement a day, and each time passed from one to two drams of bright red blood, without pain or rectal tenesmus. Each bowel movement was dark colored, but never tarry, and normal in consistence. There was no increase or decrease in the number of bowel movements, and no blood was passed between bowel movements. He thinks that he had a slight protrusion of the bowel at the anus when straining at stool at this time.

Hemorrhages from the bowel gradually increased in quantity, but not in frequency, until July of this year (1908). For four years preceding that time he had a feeling of discomfort in the lower part of the bowel when on his feet a great deal, but never amounting to real pain. During the last six months he has been somewhat constipated and he has had to resort to the use of cathartics.

In July of this year he began to have pain in the lower bowel and anal tenesmus, with frequent desire to go to stool, but only one to two bowel movements a day. With each bowel movement he passed from two to four drams of bright-red blood. When he had the desire to evacuate his bowel, he would sometimes strain a little and pass from one to two drams of bright-red blood without any feces. Sometimes, too, on rising in the morning he noticed that the bed-sheet or his nightshirt would be slightly blood-stained. He never passed any blood-clots.

In August of this year the condition became so bad that he

had to discontinue active business. At times he was so weak that he had to remain in bed for two or three days at a time. He was nauseated, dizzy, and very weak—as he thought, from the loss of blood. Three weeks ago he took a Seidlitz powder, and about five minutes later he was seized with very severe griping pains in the lower part of the abdomen. The pains seemed to begin on each side, beneath the costal arch, and radiated downward and inward toward the pelvis. The pain seemed to be paroxysmal, lasting three or four seconds, and the paroxysms came on every five minutes to one-half hour for the next forty-eight hours, suggesting mechanic ileus. He was slightly nauseated, but did not vomit. He passed blood simultaneously with each spasm of pain, but does not know how much; the stools were never slimy. He was ordered to bed by his physician, who said he had a temperature two degrees below normal, and gave him paregoric and bismuth by mouth.

After this he had a constant desire to go to stool, and would pass from two to three drams of bright-red blood each time he went. The doctor ordered normal salt-water enemas and gave him strychnin by mouth, with the result that he was relieved of the tenesmus. He remained in bed until he started on his journey to this hospital. Last Saturday he took a Seidlitz powder and had a slight attack of pain in the lower part of the abdomen, but no bowel movement and no hemorrhage. He has constant nausea, but never vomits; does not cough, and has no night-sweats; no symptoms referable to the gall-bladder; no cardiovascular symptoms. He has to get up to urinate two or three times a night, but he has no other urinary symptoms. He does not know whether he has lost in weight.

EXAMINATION.—A divided Seidlitz powder was given him here in the hospital to determine the size of the stomach and retaining power of the pylorus. The gas went through in one minute and twenty seconds, low tension; no bowel movement and no hemorrhage followed. His stomach is a little lower than normal. Examination of the feces made October 20, 1908, showed no blood and no protozoa. A Gram-positive bacillus resembling the Oppler-Boas bacillus was found.

Blood examination made October 18, 1908, showed 9800 leukocytes. Differential count showed: Lymphocytes, 8 per cent.; large mononuclear and transitionals, 1 per cent.; neutrophils, 91 per cent.; eosinophils, none. Another examination of the blood made October 18, 1908, showed 21,600 leukocytes; hemoglobin, 60 per cent. He looked like a patient suffering from malignant disease or pernicious anemia.

Examination of the urine made October 18, 1908, showed: No albumin; no sugar; urea, 1.0; no casts; no red blood-cells; no crystals; few epithelial cells; specific gravity, 1018; reaction acid. Examination of the urine made October 19, 1908, showed: A trace of albumin; no sugar; urea, 1.2; few granular casts; no red blood-corpuscles; few epithelial cells; no crystals; white blood-cells, eight to ten to a field; specific gravity, 1015; reaction acid. Examination of the urine made October 30, 1908, showed: No albumin; no sugar; urea, 2.6; specific gravity, 1028; acid reaction.

The proctoscopic examination, as in the case just cited, was negative.

When the diagnosis was cleared, the treatment was easily and effectively carried out, as is so often the case. *The mistake so commonly made is treating the patient before the diagnosis is made.*

In this case the sphincter was dilated, the bleeding point was found as a slit one-sixteenth of an inch in one of the hemorrhoids; the hemorrhoid was caught in a clamp and cauterized with the Paquelin cautery. The patient made a good recovery, and has remained well since.

In these cases one should never attempt to make a diagnosis without having made a personal examination of the stool; or, better, see the patient in the act of defecation, because that tells the story in an instant as to where the blood is coming from. I got high up in the bowel in this case with the fourteen-inch proctoscope and found nothing. Just inside the anus I found a little blood, notwithstanding that he passed a lot of blood immediately after coming into the hospital a few hours previous, which the nurse did not save.

Examining with the proctoscope there was nothing to be seen because it was a vein that was bleeding through a very small slit which was visible only when the patient strained and blood squirted from it. When the patient stops straining there is nothing to see, because the slit in the vein is closed. We do not know whether this man will bleed any more because he has not enough blood to favor coagulation. Last night we put him on proctéal salines with calcium chlorid to fill his blood-vessels for the operation, one of the good things to give in hemorrhages of this kind. We will operate on this man under gas anesthesia, as we dare not risk using ether, his condition is so very low. He was somewhat delirious last night. I never before saw a patient with only 12 per cent. hemoglobin, not even in pernicious anemia.

With the patient in the lithotomy position the anus is thoroughly stretched with the thumbs, and immediately the hemorrhoids protrude from the anus. Note how large they are and how pale the rectal mucosa is; it is almost white. Examining these masses,—there are three of them,—we find a thin, watery, sanguinolent fluid oozing from the upper surface of one of them. On close inspection I find that it is a slit in a vein, not longer than one-sixteenth of an inch. That is where he has been bleeding from. Ordinarily, I would clamp that hemorrhoid, cut it off, and cauterize the stump, but I do not dare to do that in this case because of the exsanguinated condition of the patient. See the color of that blood—it is straw colored! I am not even going to remove his external hemorrhoids. I will compress the base of each mass with the angiotribe and tie a heavy silk ligature into the crease. These ligated hemorrhoids would become edematous below the ligature if they had not been crushed with the angiotribe before ligating. Never transfix a hemorrhoid. There is practically not one raw surface on these hemorrhoids. That is all we will do in this case. The surface is covered with vaselin. A plain dressing will be applied and held in place with a T-binder.

Let the record show that there was a dilated vein on the surface of the smallest of the hemorrhoids; that that was the vein

which had the slit in it; that the slit was not more than one-sixteenth of an inch in length.

He will be given a quarter of a grain of morphin and one-sixtieth grain of atropin. He will not have salines now, because his blood is so thin. We will elevate the foot of the bed and keep him perfectly quiet, and possibly we can get coagulation of his blood early in the vein before the ligature is sloughed off. We will keep his bowels bound up for ten days to favor healing in the rectum.

Examinations of the blood in this case made after the operation were as follows: January 25: Hemoglobin, 32 per cent. January 31: Hemoglobin, 35 per cent. February 3: Hemoglobin, 40 per cent.; reds, 3,200,000. February 11: Hemoglobin, 45 per cent.: reds, 3,264,000. February 28: Hemoglobin, 62 per cent.

Now let us take up the history of the second case.

Patient, Mrs. T., aged twenty-nine, entered the hospital January 9, 1914, because of severe bleeding from the bowel.

In November, 1911, she first noticed blood in the stools. She has at no time been constipated. After each stool there is a feeling of incomplete evacuation of the rectum. At times it is painful for her to sit down on a chair. When there is bleeding, she has less soreness. The bleeding comes on in attacks lasting one-half hour after stools. These attacks continue for a few days, then for a day or two there is no apparent bleeding. Walking increases the bleeding. During the past two years the patient has had a tendency to strain while at stool. Patient had a severe hemorrhage this morning.

EXAMINATION (PROCTOSCOPIC), JANUARY 10, 1914: There is no blood in the rectum for a distance of eight inches from the anus, despite the hemorrhage this morning; it evidently came from the anal margin. There is a small ulcer on the posterior margin of the anus—an erosion of a hemorrhoid. The anemia is secondary.

EXAMINATION OF BLOOD: Leukocytes, 5200; erythrocytes, 2,560,000; hemoglobin, 32 per cent.; blood-pressure, 116. Differential leukocyte count: Lymphocytes, 15 per cent.; large

mononuclears and transitionals, 8 per cent.; polymorphonuclear neutrophils, 74 per cent.; eosinophils, 2 per cent.; basophils, 1 per cent. Differential erythrocyte count: Poikilocytes, about 40 per cent. of field; megalocytes, few in every field (nearly 80); microcytes (stained), denoting secondary anemia, quite a number in every field; normoblasts, none; megaloblasts, none; microblasts, none; polychromatophilia, sparse.

DR. MURPHY: This case is a very interesting one, and I will ask Dr. Mix to discuss the blood-picture and the diagnosis from a differential standpoint in detail, as errors in diagnosis in primary and secondary anemia many times cause the loss of life which might be saved.

DR. MIX: From the point of view of the blood-picture, the case operated upon today and the case operated upon yesterday are exactly alike. Both of them show an intense anemia secondary to bleeding hemorrhoids. Strictly speaking, the bleeding could not be said to be from hemorrhoids proper, though large hemorrhoids were present in both, but rather from minute bleeding points in the hemorrhoidal veins close to the anal margin. In the case of the man operated upon yesterday, the examination of the blood by the intern shows that the hemoglobin was reduced to 12 per cent. Such a degree of reduction scarcely seems possible when correlated with the reduction of the red blood-corpuscles, which are reduced to 1,728,000. This reading of 12 per cent. was made with the Tallqvist paper determination of the hemoglobin.

In estimating these cases from the point of view of operation and treatment, the main thing is the differential diagnosis between primary pernicious and secondary anemia. In primary pernicious anemia the blood-picture in the average case may be something like this: The hemoglobin will be perhaps 40 per cent.; the red blood-cells will perhaps run to about 1,200,000, with the result that the color index is greater than unity. Expressed in another way, the hemoglobin is relatively too high in amount for the number of erythrocytes. You can always estimate the color index by making an ordinary proportion as follows: 100 per cent.

of hemoglobin is to 5,500,000 reds as x per cent. of hemoglobin is to 1,200,000 reds.

$$100 \text{ per cent.} : 5,500,000 : x \text{ per cent.} : 1,200,000$$

Multiplying the means by the extremes, we have $5,500,000 x$ equals 120,000,000. X equals 120,000,000 divided by 5,500,000, or 21.8 per cent. This, when interpreted, means that 1,200,000 reds ought to contain 21.8 per cent. hemoglobin. As a matter of fact, in our hypothetic case of pernicious anemia the amount of hemoglobin which they actually did contain was 40 per cent. The color index is obtained by dividing the hemoglobin found by the hemoglobin which ought theoretically to be present; and, therefore, dividing 40 per cent., the hemoglobin actually present, by 21.8 per cent., the hemoglobin which ought to be present, we find the color index to be $\frac{40}{21.8}$, or 1.8.

Practically, all cases of pernicious anemia show a color index above unity. It is true that there are some borderline cases occasionally found where the color index is very close to unity, and where considerable judgment is necessary in determining whether the case is a primary or a secondary anemia. In general, however, the cases of primary pernicious anemia without exception show a color index above unity.

There is another very important differentiation between primary pernicious anemia and secondary anemia, based upon the differential red blood-count. In cases of pernicious anemia it will be found, on examining the blood, that the corpuscles are of various sizes and shapes; a few of them are too small in diameter; many of them are very much too large in diameter, and most all are irregular in outline. The corpuscles having the irregular outline are called poikilocytes. Those which are smaller than normal are called microcytes, and those which are larger than normal are known as macrocytes. It is quite obvious that if the blood of a pernicious anemia contains much more hemoglobin than the number of red corpuscles would normally warrant, that many of the red corpuscles must be carrying a very much heavier load of hemoglobin than normal. These are the

macrocytes, *and they number in cases of pernicious anemia 40 per cent. or more.*

There is a practical point in the differentiation of the red cells which must be called to your attention, and that is that in making it one must, to quite an extent, rely upon his mental picture of the normal size and appearance of the red corpuscles. In counting three or four hundred red corpuscles, there might be some difference of opinion between the different observers as to just what corpuscles ought to be called macrocytes. Care, however, will usually lead to the discovery that the macrocytes exceed 40 per cent.

Once more, why do you have 40 per cent. or more of macrocytes? To repeat, a macrocyte is a large cell containing more than the normal amount of hemoglobin. Then in the blood of a patient having a color index of 1.8 there necessarily must be a large number of macrocytes present in order to accommodate the increased load of hemoglobin.

The differential red count also shows at times nucleated red cells. These are usually named, in accordance with their size, microblasts, normoblasts, and megaloblasts, the terms defining themselves. The nucleated reds are very rarely microblasts. Usually they are normoblasts and occasionally megaloblasts. Another characteristic in pernicious anemia of the red cells is the fact that many of them stain in a peculiar way, being basophilic.

In cases of pernicious anemia *the white corpuscles also furnish a means for differential diagnosis.* Usually in pernicious anemia the white count runs from 4000 to 4500. The cases never show an apparent leukocytosis, though as a matter of fact a relative leukocytosis is always present; expressed in another way, not an absolute leukocytosis, but a characteristic reduction of the total number of leukocytes. That reduction, however, relatively is not extreme, as is the reduction of the red blood-corpuscles. This may be amplified in explanation by another statement. If, for example, the red blood-cells are reduced from 5,500,000 to one-fourth that amount, then the white blood-cells are not reduced from the normal 7000 to one-fourth that amount. Such

a proportionate reduction never takes place. There is then a relative leukocytosis present, but an absolute leukopenia.

In making the *differential examination* of the white blood-corpuscles, what do we find in the ordinary individual in health? The lymphocytes should be approximately 20 per cent. to 24 per cent.; the polymorphonuclears should run 65 per cent. to 70 per cent. In pernicious anemia these figures are changed. The lymphocytes are always increased, frequently running to 40 per cent., and the polymorphonuclears are correspondingly reduced, frequently between 45 per cent. and 50 per cent.; the large mononuclears which run about 5 per cent. to 8 per cent. in normal blood will also be frequently found to be increased in the blood of pernicious anemia.

Let us now take up the blood-picture of the patient who is going to be operated on this morning. We find these figures: the hemoglobin is 32 per cent.; the red cells are 2,560,000. Let us calculate the color index. One hundred per cent. hemoglobin is to 5,500,000 reds as x per cent. hemoglobin is to 2,560,000 reds.

$$100 \text{ per cent.} : 5,500,000 :: x \text{ per cent.} : 2,560,000$$

Multiplying the means by the extremes as before, we have $5,500,000 x$ equals 256,000,000. X equals 256,000,000 divided by 5,500,000, or 46 per cent. In other words, having 256,000,000 reds, the patient should have 46 per cent. of hemoglobin. She has, however, actually 32 per cent. Dividing the amount of hemoglobin which she actually has by the hemoglobin which she should have, according to the rule which we followed in the previous case, we find the color index to be 0.7. In other words, in this particular patient we have a color index that is below unity instead of being above unity. The conclusion is inevitable that she is suffering from a secondary and not from a pernicious anemia.

Furthermore, when we note the differential count of the red blood-cells we find that the macrocytes are recorded as merely a few in every field. We find that the microcytes which usually predominate in secondary anemia show quite a number in every field. Furthermore, we note the absence of normoblasts, megaloblasts, and microblasts; that is to say, there are no nucleated red cells present.

Let us now turn to the differential count of the white blood-cells. We find that of the lymphocytes there are only 15 per cent. In pernicious anemia they usually range from 30 per cent. to 40 per cent. We find the polymorphonuclears 74 per cent. In pernicious anemia these usually run 50 per cent. or less. In other words, after making the examination of the white blood-cells in this patient we are led to the conclusion that they present the picture of a secondary anemia and that they differ radically from the percentages which obtain in a primary pernicious anemia.

That a severe secondary anemia may sometimes temporarily assume the picture of a primary pernicious anemia if it is not taken care of I do not deny. I maintain, however, that in the vast majority of cases the differential diagnosis between a primary pernicious anemia and a grave secondary anemia is usually possible if attention is paid first to the color index; second, to the percentage of macrocytes; third, to the percentage of lymphocytes; fourth, to the percentage of polymorphonuclear cells.

The blood-picture in the first case operated upon yesterday is also one of a very grave secondary anemia. The hemoglobin is recorded as 12 per cent., the red blood-cells being 1,728,000. The color index, which may be calculated out by the rule which I have twice illustrated upon the blackboard, will be found to be 0.38. This is the lowest color index I have ever seen, and the reading of 12 per cent. made by the Tallqvist paper may really be too low. It surely, however, is as low as 20 per cent., so that the color index is surely as low as 0.64. This latter is also a very low color index, but one that we not infrequently meet with.

We find on examination of the white blood-cells that the number of leukocytes per cubic millimeter is 4800. The differential count shows the lymphocytes to be 18 per cent., a very slight reduction; the polymorphonuclear cells to be 76 per cent., a very slight increase above the normal. We find the large mononuclear and transitional cells to be 4 per cent., a slight reduction. In other words, the differential leukocytic count shows the changes of a secondary anemia and not those of a primary pernicious anemia, the latter showing an increase in the lymphocytes

and a reduction in the polymorphonuclears. In the differential count of the red cells we note that only several megalocytes are found, whereas in a pernicious anemia we would find that they are above 40 per cent. in amount.

A point in the examination of this patient's blood, which occasionally proves a stumbling-block to some in the differential diagnosis between primary pernicious and secondary anemia, is the fact that three normoblasts were found. Normoblasts, or nucleated red cells of normal dimensions, are occasionally found in severe cases of secondary anemia. In themselves alone they are never a sure indication of primary pernicious anemia. They occur in both blood-pictures. In this particular case they serve merely to indicate the intense degree of secondary anemia and to prove that the bone-marrow has been making the greatest possible effort to restore to the blood-stream the missing corpuscles. In nature's endeavor, a few unfinished ones have been supplied.

There is another wide and fundamental difference between primary pernicious and secondary anemia. The former is a part of a general intoxication or infection and frequently involves the central nervous system. The latter is merely the result either of the intense action of hemolysins or of a direct blood loss. In the latter, therefore, changes in the central nervous system are not encountered except as accidental findings. In the case of pernicious anemia the physical signs depending upon an involvement of the spinal cord will be found to be present in about 60 per cent. of the cases. Formerly it used to be said in the text-books that only a few of the cases of pernicious anemia showed an involvement of the cord. Now we know, however, by reason of the numerous and careful observations which have been made in these cases by competent observers, that more than half of them show cord changes. Relatively speaking, these cord changes result in two types of cases, the ataxic type and the ataxic paraplegic type.

In the *ataxic* type the symptoms are very much like those of locomotor ataxia except that the Argyll-Robertson pupil is lacking. The signs of the two diseases are exactly alike, however, in

the following respects: both show absence of the knee-jerks; absence of the Achilles jerks; delayed conductivity of sensation; loss of muscle and joint sense or its reduction; loss of tactile sensation. In addition, depending upon the involvement of the muscle and joint sense, there may be a certain amount of ataxia. There is the same cotton-sole sensation on the bottoms of the feet in both conditions, depending upon the tactile loss present.

Whenever you have a case of severe primary pernicious anemia, therefore, always tap the patellar tendons to find out whether the knee-jerks are present or absent. Never omit to have the patient kneel on a chair with his feet out behind him so that you may properly examine the Achilles jerks. This you may do by tapping the Achilles tendon with your percussion hammer. The next important test is to determine whether the patient is conscious of which way you move this big toe while his eyes are closed. You will be astonished, if you make this examination of each case of pernicious anemia which you come across, to see the large number who will not be cognizant of the direction in which their big toes are being moved. In some instances you will find it possible to move the whole foot at the ankle-joint without the patient being aware that the foot is being moved or in what direction it is being moved.

Further careful examination of the sensation will show small areas of tactile loss in the foot and leg just exactly comparable to such tactile losses as occur in locomotor ataxia. These losses of the muscle and joint sense just mentioned mean an involvement of the columns of Goll and Burdach. If the tactile sense is also affected, you may be sure that the columns of Goll and Burdach are affected from the posterior fissure to the central commissure, because many of these tactile fibers run in the base of the columns of Goll and Burdach close to the gray matter not far from Clarke's column. Examination of the cranial nerves, however, shows none of them to be affected. This is contrary to the rule in tabes, where an early involvement of the third nerve, either in the form of a ptosis or a strabismus, is an early sign. Furthermore, in pernicious anemia the Argyll-Robertson pulse is not present. Lastly, and very important, there are no pupil-

lary inequality and no irregularity of pupillary outline to indicate an antecedent syphilis, such as we often find on the examination of the pupils in cases of locomotor ataxia.

I do not mean to affirm that pernicious anemia may not be at times upon a luetic basis. There is reason to believe, from the recent success of salvarsan, as first proposed by Byron Bramwell, that not a few cases of primary pernicious anemia are upon a luetic basis, and these are, of course, made worse by the luetic taint. If lues be present in a patient showing a primary pernicious anemia and also the cord symptoms, it will be exceptionally difficult, if not impossible, for the neurologist to state the exact etiology of the cord symptoms present, provided the pupils are unchanged in their reflexes.

It will be recalled that I said that the nerve symptoms of pernicious anemia are confined to one of two types, either the tabetic type or the *ataxic paraplegic* type. In those cases of pernicious anemia which are in the latter category there is a direct involvement both of the posterior and of the lateral columns; that is to say, of the columns of Goll and Burdach, carrying the muscle and joint sense fibers and the pyramidal tracts carrying the motor impulses from the brain to the anterior horn cells. As a result we find, just as we find in ataxic paraplegia, that the knee-jerks are very much increased in action; that the ankle-jerks are also increased. We may even find quite a degree of spasticity, depending upon this involvement, and some paresis of the lower extremities. If the cervical region of the cord is involved, the disturbance may appear in the upper extremities, as in a case under my care during the month of November.

We find also, as in the tabetic type, some loss of muscle and joint sense betrayed by the lack of knowledge on the part of the patient of the way in which his big toe is being moved, and also of the tactile sense, betrayed by his lack of sensation when the cutaneous areas over the dorsum of the foot or toes are very gently stroked.

These cord changes, either of the tactile or ataxic paraplegic types, are to be expected in cases of pernicious anemia, sooner or later. In not a few cases the cord changes usher in the disease.

I have on two occasions made the diagnosis of pernicious anemia before examining the blood, merely from the presence of the nerve symptoms. They were so characteristic that I knew the pernicious anemia must be present, although the blood examination had not been made. One of these cases was a maiden lady, about fifty years of age, whom I saw in consultation with her physician. He invited me to confirm a diagnosis of ataxic paraplegia. I was able to confirm this diagnosis, but also to assure him that the etiology was a pernicious anemia. Examination of the blood showed 70 per cent. hemoglobin; about 4,500,000 reds. The pernicious anemia subsequently became more pronounced and ultimately caused the death of the patient.

The fact that the disease which underlies the cord changes and the blood changes of pernicious anemia may attack either cord or blood first is the basis of the suggestion that there is some constitutional intoxication or infection present in the body with an especial predilection for blood-producing and nerve tissues.

Cases of severe secondary anemia do not show such cord changes. The knee-jerks and ankle-jerks are not changed. There are no indications of an involvement of the columns of Goll and Burdach. There are no tactile losses. There is no ataxia and no paresis of the lower extremities. There may be weakness, but the weakness is general, involving the whole of the body equally, being consequent merely upon the blood reduction. There will be no weakness which will be found to be due to an involvement of a particular portion of the central nervous system.

It will be seen then that the differential diagnosis between primary pernicious and secondary anemia is in the great majority of cases comparatively simple. It depends very greatly upon a careful examination of the blood and of the central nervous system. In pernicious anemia the blood will always show a color index above unity; ordinarily a large number of megalocytes, not infrequently nucleated reds, an increase in the number of lymphocytes, and a decrease in the number of polymorphonuclear cells. The central nervous system, furthermore, will be found to be affected in more than half of the cases.

DR. MURPHY: You will note the intense anemia here. She

has the color of a pernicious anemia patient. In the first proctoscopic examination we were unable to determine anything, except that there was a little blood in the rectum at its lower margin, but none in the upper bowel, using the eight-inch proctoscope. Yesterday we made a second proctoscopic examination and we were able, without any trouble at all, to demonstrate the slit in the vein from which the blood came. It was no larger than the lead in a lead-pencil.

We operated on this woman just as we did in the first case and easily found the slit in the vein.

The blood examinations in this case after operation were as follows: January 25: Hemoglobin, 30 per cent.; reds, 3,250,000. January 31: Hemoglobin, 30 per cent.; reds, 3,300,000; whites, 9000. February 11: Hemoglobin, 30 per cent.; reds, 3,264,000. February 15: Hemoglobin, 30 per cent. +; reds, 3,360,000.

Additional report in this case made April 5, 1914: Number of leukocytes per c.mm., 6200. Number of erythrocytes per c.mm., 3,744,000. Percentage of hemoglobin, 70. Patient still has some ataxia of the lower limbs, but is steadily improving.

Never transfix a hemorrhoid. If you will look over the literature, you will find that in many of the cases in which there was trouble after ligature operations were those in which the hemorrhoid had been transfixed. I recall a case I saw in consultation. The patient was a young man whose hemorrhoids had been operated on by the transfixion method four weeks previously. He got along in the usual way, went home on the sixth day, and suddenly, on the seventh day, developed irregular recurrent chills and fever, followed by a mild icterus. He had a little discomfort in his abdomen, and a deep tenderness, which was not, however, a real pain; but he had an enlargement of the liver and of the spleen. There was no diarrhea and no tympany. I examined him, went into the details of the operation and learned that he had his hemorrhoids ligated by transfixion. The liver and spleen were both sensitive to fist percussion. He had a peculiar chill and temperature. A phenomenon typical of intravenous or cholangic infection is chill and temperature up to 104° or 105° F., and in a few hours return to normal; on the same day

a repetition of it—then freedom from chills and slight temperature for several days and then again a repetition of same angular temperature with complete intermission. It was certain in my mind that he had a pylephlebitis, namely, an infection of his portal vein, due to the fact that in operating on him the doctor transfixes his hemorrhoidal vein. This undoubtedly started the infection, and the ligature allowed the admission of micro-organisms into the vein from the inferior hemorrhoidal; it progressed to the middle and then the superior hemorrhoidal and the inferior mesenteric and was then in the portal vein.

The autopsy was made by Dr. H. G. Wells, of the University of Chicago. He just opened that thrombosed hemorrhoidal vein from the transfixion and followed straight up to the portal circulation. There were abscesses in the portal vein, abscesses in the liver, and abscesses in the spleen, showing that an infection had occurred, because the needle, as is always the case in this operation, was pushed through the vein.

Remember that the inferior hemorrhoidal vein collects blood from the anus and empties into the internal pudic vein, a branch of the internal iliac; an anastomosis with the middle hemorrhoidal vein helps to form the hemorrhoidal plexus, which empties directly into the internal iliac vein, but also anastomoses with the superior hemorrhoidal vein, which is a direct branch of the inferior mesenteric vein and inosculates with the middle hemorrhoidal branches of the internal iliac and thus establishes a communication between the portal and the general venous system. The portal vein is formed by the superior and inferior mesenteric, the splenic, gastric, and cystic veins. The course of the infection in the case I cited can thus be traced easily from the middle or superior hemorrhoidal into the portal vein, the liver, and the spleen.

In looking through the literature on this subject you will find that the best article was written by one of the members of the staff of Guy's or St. Bartholomew's Hospital. It is included in the report of the hospital. He cited cases of pylephlebitis that occurred from ulcers in the stomach, ulcers of the intestine, ulcers in the lower and upper rectum, infections of and around the ap-

pendix. It is one of the best papers I have read and cites many examples of each of the conditions that may cause a pyelephlebitis.

Still another case today which fits into this category by accentuating the necessity of early diagnosis was the following, first seen a few days after we operated on the second patient mentioned above.

HISTORY.—Patient, male, aged thirty-four, entered the hospital January 14, 1914, with the following history: For the past four years has had chronic constipation, preceded for many years by a rectal tenesmus. He frequently has a sensation of incomplete evacuation of the rectum immediately after stool. Occasionally he strains at stool in his endeavor to empty the rectum, and there are frequent periods of bleeding, but each time this has been stopped by a suppository given him by his doctor. There are times when it causes some distress to sit down.

Twelve years ago he acquired an inguinal hernia from lifting heavy boxes and for this he began to wear a truss. Paraffin was injected into the canal six years ago. During the past two years this truss caused him pain by pressing the lumps of paraffin against the underlying soft tissues.

DR. MURPHY: This patient had a hernia which was treated by the paraffin injection plan. I do not know how much of a hernia there was in the beginning, but he now has a mass of paraffin here, around which connective tissue has formed. It must be removed because it is causing him pain. We make our incision as for a herniotomy, because we may have to deal with one before we get through. After dividing the sac see that paraffin, and note the firm, thick capsule of connective tissue around it, and then remember that paraffin has been suggested as a treatment for the prevention of ankylosis and for the prevention of peritoneal adhesions. Just a few weeks ago we had a case of extensive intestinal adhesions which had been treated by paraffin, and those of you who were here at that time will remember what extensive or universal and firm adhesions had formed around the liquid paraffin, showing that it increases rather than lessens the adhesions.

The hernial opening is now closed—I do not know how much

of a hernia he had originally. There is the aponeurosis of the external oblique. I want to get that in shape and put in a few stitches to give that external ring more support, which it needs. He does not need to have a radical operation for hernia at all, because he has no hernial sac.

Now we will place the patient in the lithotomy position and operate on his hemorrhoids. These are very inoffensive hemorrhoids on the outside—they are only little skin-tags. I will stretch his sphincter, and as I do so a number of small internal hemorrhoids come into view. We grasp each one at its tip with a hemostat, incise the mucocutaneous tissue at its base all the way around on the skin side, strip it down, and put on a hemorrhoidal clamp in the crease, and with the curved scissors the hemorrhoid is then sliced off the top of the clamp and the stump is cauterized with the Paquelin cautery. That is the ideal operation in these cases. When the clamp is removed, the cuff of mucosa falls in over the cauterized base of the hemorrhoid, covering it over completely so that there is little raw surface exposed.

All these cases show that when you have a case of blood coming from the bowel, find out what is the matter. Make a proctoscopic examination. If you do not see the bleeding point exposed, make the patient strain and see where the blood comes out. Do not give suppositories to check the bleeding. Do not give opium or morphin to cure the lesion, because it will fail to do so. Find the bleeding point, and then carry out the indicated treatment.

Now just a word as to another operation employed in cases of hemorrhoids.

Excision with ligation is a clamping of the hemorrhoidal tumors at their base, ligation at this point, and amputation. Much pain and reflex disturbance can be avoided by splitting the cutaneous or mucous surface and peeling it backward above the line of ligation. The vitality of the stump at the point of ligation should be destroyed completely by the crushing effect of the angiotribe; this diminishes the subsequent pain and destroys the reflex.

A recumbent position is favorable for convalescence; it is not indispensable, however. Healing is slow and free from pain. Fistulæ and fissures do not appear after the cautery operation. Stricture is a sequel of the removal of too much cutaneous or submucous fibrous tissue, and is one of the most unpleasant sequences; it should be operated early. True recurrences after an efficient operation are very uncommon. The little tags of skin are frequently erroneously considered by the patients as a return of the hemorrhoids.

MURPHY'S CLINICAL TALKS ON SURGICAL AND GENERAL DIAGNOSIS

TALK ON THE IMPORTANCE FOR PROFESSIONAL SUCCESS OF SKILL IN DIAGNOSIS AND PROGNOSIS. HOW SURGERY SHOULD BE STUDIED

FRIDAY, MAY 22, 1914

To Dr. Murphy: We, the members of the Senior Class of Northwestern University Medical School, on this our last clinic day, wish to take this form of expressing our sincere appreciation to you and your staff for your regularity, punctuality, and faithful and efficient service in giving to us such a thorough and systematic course of instruction as we have received during this past school year.

L. J. PALMER, *President*

GENTLEMAN: "*What's the matter with him?*"

THE CLASS: "*He's all right.*"

DR. MURPHY: Members of the Senior Class: I greatly appreciate your generous approval embodied in these resolutions. You probably do not realize what it is to a teacher thus to have the confidence and respect of his class. You probably do not realize what a stimulus it is to a teacher when talking to you to see that your eyes and minds are riveted on the things he is endeavoring to demonstrate—neither do you realize what an enormous responsibility a teacher has if he does his duty to his class. His primary responsibility is to you; but his ultimate responsibility is enormously greater, and is farther reaching than you, perhaps, at this time appreciate; for it is not only through that which he imparts to you of actual information, but also through training you to acquire further learning, that he does his duty to you and to the people of our commonwealth, whose lives and health will be intrusted to your care.

You can see, therefore, that it is not merely a matter of lessons or of the teacher. It is a matter of preparing you to render efficient service to those who intrust themselves to you.

Not only that, it also means preparing you so that you in turn may prepare another generation for this great work. It means instilling into you not only a detailed knowledge of medical facts, but also, and much more vital to your future usefulness, the desire for still broader training, together with the proper guidance for its pursuit. Imparting a knowledge of the route which one must follow to secure scientific advancement and honorable standing among one's brother practitioners and fellow-men is the most important chapter in the work of a teacher of medicine who feels a moral responsibility to his students.

I am happy to say that during this entire course I have had, I think, a larger percentage of the members of the class concentrating on the things we have been doing in the arena than I have observed in a class before. It seems to me that you all have felt a desire to be in harmony with the thoughts in your teacher's mind in his demonstrations. I admire the spirit with which you daily have come into the arena and stood the quiz ordeal, knowing that it was for your best interests and for the best interests of the members of the class that these sharp-pointed questions were put. I am sure that no man in leaving this arena will feel that in these cross-questionings we have had anything in view but a desire to set the greatest possible number of members of the class to thinking along proper lines, and that these quizzes were, therefore, entirely impersonal.

I believe that this question and answer method of discussing clinical subjects with members of the class is the best way to train you to think for yourselves. It is not so much the information I give you which is so important, as it is the methods of seeking information which I try to impart to you by the questions I ask. Remember the child's education—that is the key to my system. The child's education comes by asking questions—asking questions; and *your* education will not continue to grow unless you keep asking yourselves pertinent questions and unless you devote some time of every day to medical authorities to hunt down the answers to these questions. If you sit in your library and dream about the answers, you will not accomplish much; but if you formulate a system of asking yourself questions about

your cases and then have that little library—small is all it need be, but recent as you can have it—and put a constant appeal for light to some competent authority on the subject, and if, when you read and study, you do not say, “I read so many pages to-day,” but, “I attained so many new points of information”—*then* you will be making mental progress. When you answer questions in your line of investigation by looking up the proper authority, that knowledge becomes yours, because it is knowledge practically applied. If you merely read in an indifferent way, that knowledge remains the man’s who wrote the book and does not become yours. It is the system of asking questions about actual cases and getting the answers—now remember, *getting the answers*—that will enable you to attain the skill, knowledge, foresight, and, finally, diagnostic ability, which I believe is the highest accomplishment attainable in medicine.

You will note that in the student clinics we have devoted little or no time to operations. Why? Because students remember little of the details of these operations. If you wish to study operations, you can do it from a text-book, in which you get the information presented to you in better form than it can be presented by an operator as he goes along with his work. But diagnosis you must learn by a system of personal investigation of actual patients, and if you have such a system clearly in your minds, you will finally become diagnosticians and you will be capable of doing ably that thing on account of which the profession suffers most, because it lacks it most; that thing through deficiency in which we have lost more people’s confidence than through any other one, namely, diagnosis. People do not expect us to cure them of all their ills, because they all expect to die sooner or later. They all recognize in life so dangerous an undertaking that none of them expect to get through it alive. But they do expect us, when they appeal to us for aid, to devote sufficient time, skill, and care to arrive at a correct diagnosis of their condition; and if we do that, our treatment can scarcely be faulty. Success rests almost entirely in arriving at a diagnosis. There are fairly clean-cut and accurate lines of treatment laid down in the text-books, with the exception of some individual

cases, and the text-books will take care pretty well of that phase of your work. The diagnosis, however, must be all yours. You cannot get it out of one book or two books or three books. You must get it by repeated analysis of case after case, by constant study, by continual practice in physical examination in going over and over those portions of the body where you do not much expect to find a lesion in the individual case, but where you examine to familiarize yourself thereby with normal conditions; and often you will also be rewarded by finding pathology when you were not looking for it and when you did not expect it from the patient's story.

When I say that the failure to diagnose correctly is what in the future we shall be held accountable for—and rightly so—let me quote from the *New York Times* the report of the committee which has been investigating the service of some of the New York dispensaries and hospitals. That report states that 47 per cent. of all the cases that come to the dead-house table in New York City,—and remember that it is superlative disease when it is brought to autopsy,—47 per cent. of those cases that come to autopsy table have errors in diagnosis. That is a sad comment on the work of our profession. Remember, when you get such a colossal percentage of error in those grave diseases that it does not actually represent 47 per cent. of incapacity on our part to diagnose. That enormous percentage of error represents largely the indifference of the diagnostician and his indolence—not his incapacity. There is no profession to which a man can apply himself which requires such continuous, strenuous, and never-ending labor as medicine—none. The important advances keep coming so fast and in such numbers that, unless one is alert and studying every minute, it is but a short time until he drops into the past tense, so far as medical knowledge is concerned.

In your arrangement of your future work from a surgical standpoint, may I just add a few more suggestions? In the practice of surgery your act may become a menace to the patient's life; but your act is no greater a danger to the patient than your negligence in not making a diagnosis and not acting promptly

on it when made. The physician following internal medicine as a specialty who fails to make a thorough examination, also fails to make correct diagnosis. Such an internist is just as great or even greater a danger to the patient from a life-saving standpoint than the surgeon who does careless work. It is not so noticeable, perhaps, in the internist, and the patient's death is not so flaunted in his face, as it is after the surgeon has performed a laparotomy and the crape is on the patient's door because of fatal hemorrhage on the table; but the number of lives lost by failure to diagnose and by procrastination in "working-up" the case properly is enormously greater—thousands per cent. greater—than are the deaths due to incompetent surgical acts. What do I mean by that statement? I refer to the doctor who sees a case of carcinoma when it is a small nodule in the breast; the practitioner who sees the epithelioma on the lip when it is a small sore; the physician who sees his appendicitis case in the first colic, his case of intestinal obstruction when the patient is having the first emesis. The internist who finds his patient suffering from an acute virulent infection and does not arrive at a diagnosis then and there or does not possess sufficient mental force to demand prompt action, sacrifices enormously more lives than does the surgeon. Every doctor of mature experience recognizes the veracity of that statement in an instant. Take lip epithelioma, for instance. How many cases of epithelioma of the lip come to the surgeon within the first six months of the appearance of the lesion? Not 5 per cent.—not one in twenty. Think of it! Such a cancer is flaunted in the face of the doctor, it is flaunted in the face of the patient and his people, and still procrastination, procrastination, procrastination, is the order of the day until the glands in the neck become involved; and when the glands have become involved, that patient has a 76 per cent. chance for a cancer death, even if operated carefully and thoroughly, from a lesion which was originally so small and so easily removed that it would have been almost like picking a cherry to take it out. And 76 per cent. of these people must die because of this neglect.

The positiveness of your diagnosis also is an important factor

to you and to your patient. After you have made your diagnosis and proved it up as you would a problem in mathematics, then take a positive stand as to what should be done and stick to it.

Wednesday of this week, at the Illinois State Medical meeting, the subject of the management of acute appendicitis came up and there was at the meeting a man, I think he was close to sixty years of age, who reported before that large body of medical men that all his cases of appendicitis got well when treated on the expectant plan. His statements naturally were roundly refuted by his audience.

I cite that instance to show that as students we should feel great respect for the overwhelming force of numbers in medical opinion; that we have no license to rush headlong into opinions so markedly at variance with clinical and autopsy statistics. We have no right to advance and support publicly an opinion radically at variance with the general current of medical thought unless we first satisfy ourselves as to its truth by careful experimental, clinical and pathologic study and a careful study of the observations of others along the same line, as you find them recorded in the literature. That is the way in which genuine advances are made.

We have a right, on the other hand, to differ from the greatest known authorities on any particular point if we can stand forth definitely and positively and "make good" on the basis outlined above; and we *should* do so because that is the way in which all scientific advances are made. If you have not the courage to break through the lines of tradition of your time when you know and can prove them to be in error, then *you* will not be the one to make advances. But remember, that just as soon as you break through that line of tradition, you will become the target for sarcasm, hostile criticism, and actual injustice; but they are really blessings in disguise, for they are what make *men*. It is overcoming opposition which develops you and you cannot develop to your fullest strength and capacity unless you have it.

A great deal of your success depends, however, on how you meet this opposition. You must have a superlative respect for scientific truth. In seeking facts which strengthen your position

use legitimate means, legitimate statistics, and present your case in a strictly legitimate way. Then you will succeed and your success will be permanent. If you use any other means in endeavoring to strengthen your position a vicarious temporary victory will be followed by a certain ultimate defeat. You will lose your combat and be weakened permanently by the defeat. So, in advising you to adhere to well-grounded lines of practice, I am reminding you that a knowledge of the things which have been done in the past will be of great assistance to you in doing greater things in the future; but, at the same time, you must have the courage to go out and do that which has not been done before.

When taking a new step in medical procedure, you have much more to think of than your own career. Your most careful thought must be for the patient. If you perform a new operation, do not do it for the first time on the patient. Do it on the cadaver and on the dog. May I add here a few words to aid you in the development of your surgical technic? If I had to state where I acquired the greatest amount of technical knowledge, where I received the greatest benefit in the way of gaining confidence in my own operative procedures, I should say that practically all of it was acquired from operations on the dog and a smaller percentage from operations on the cadaver. Still there is no new operation of any severity or gravity, even now, that I do not perform first on the cadaver; and there is a cadaver here in the dispensary, and has been all the time that you have been in this course, just for that purpose. Where you locate you may not have cadavers at your command; but there is no place in this country situated so far from civilization and educational facilities that you cannot in a single night travel to a town where you can get the privilege of doing that operation on the cadaver. Then *do it*, because it will give you confidence in yourself when you operate on the patient and will increase your fitness to serve the best interests of the people.

The people, even when uneducated, illiterate, and entirely uninformed as to your art, are nevertheless wonderfully keen in their observation as to your knowledge; and they will form after

a short time a much more accurate opinion of what you know than you have yourself. You cannot deceive the people in great numbers for any considerable period of time. In what way do they find you out? In the first place, by your diagnosis. If your diagnosis is incorrect, what must also be incorrect? Your prognosis. Above all be honest with yourselves and then you cannot disregard the best interests of your patients. If you are uninformed do not hesitate to admit it when thereby you can obtain enlightenment. Let me cite an instance. I met a doctor in consultation one day, in a master-printer's house on Marshfield Avenue. The patient had a severe cough. It was in the early eighties and the tubercle bacillus was still relatively little known in Chicago. It was suggested to the doctor that he have the sputum examined for bacilli. He said he would do it. I met him a few days later and asked him if he examined the sputum for bacilli and he said yes. I inquired, "What staining process did you use?" He replied, "A blue lens"—and saw the bacilli! He had one of the largest practices in that neighborhood at the time; but his name is never mentioned now. He did not know a bacillus from a crowbar at the time; but that was not so much to his discredit as the fact that he did not try to learn and that he was not honest. It is impossible for such a man to make a permanent success.

For those of you who expect to follow surgery in practice let me outline a scheme of future study. First, plan to do regularly some experimental dog work, and do it with the same care that you would use on the human patient and with the same consideration for the feelings of the dog so far as pain is concerned; and after operation give the dog the same care as a patient. I have said of my dogs that they received as much nursing and attention as my patients.

Begin your experimental surgery with the arteries and veins. Why? Because the control of hemorrhage is the key to all surgical procedures. Learn to master every type of hemorrhage. You can now find in most of the text-books a chapter on the surgery of the blood-vessels. Just go and do all of these experiments yourself. Verify, by doing them in the proper way, what the other

man has done, not because you are going to use vascular suture much, but because you are going to meet arteries and veins every single day and the controlling of blood will be one of your principal functions if you are to be a success in surgery. Resect, divide, and excise portions of the aorta and reunite them. Do the same on the axillary artery and on the carotids. Make resections of veins, excise portions, and suture them. Make your knowledge of arteries and veins not a text-book knowledge, but actual experience. You can learn much more in that line on the dog than you can ever learn on the human being, because you are not at liberty to carry out for purely technical ends the procedures on the human being that you can on the dog.

After you have covered fully the management of arteries and veins, the next subject to take up is the surgery of the lung. Open the chest-wall and transplant the lung through the ribs by spreading and by resecting them. Read the technical feats which have been accomplished and do them; then do the things which have not yet been done. Remember, the surgery of the lung has been one of the slowest fields to be cultivated, chiefly on account of the bugbear of pneumothorax. This bugbear is almost a myth; and yet you all hear about it, and every student goes out of college having a horror of it. Do you know that the first removal of a lung on record was performed by a bull in Bologna nearly five hundred years ago? If a bull could extract a lung and the patient's life be saved, why should not a surgeon do it? That case is an actual historic fact. The man was gored in the chest by a bull, and, in his straining effort, when the animal also stepped on him, his lung was driven out through the opening made by the animal's horn and rested outside of the chest. The wise old doctor who saw him did not try to put the lung back in the chest. He just left it out there on the skin. What happened? Pleuritic adhesions formed, closing the wound and protecting the pleural cavity from infection. Finally the surface of the lung sloughed, the remaining stump became desiccated, the wound healed over, and the patient got well. If you look over the history of chest surgery from that time until

the present, you will not find 25 removals of the lung in five hundred years. Think of it!

It is going over the literature which will prepare you to become a good therapist. Because the cure of some particular disease is an impossibility today, remember, is no reason why it may not be the every-day practice tomorrow. You will learn in your study of pneumothorax in the animal to be masters not only of hemorrhage, but also of the other affections of the respiratory tract and of the conditions in it which can lead to death. When you get to doing surgery of the chest after faithful study of that sort on the animal, you will feel just as much at home there as you will doing your five hundredth case of interval appendicitis. There is always an "island of safety" to which one can retreat in a difficult operation and feel secure as to the patient's life; and such "islands of safety" one discovers best by dog surgery. Unless you do this experimental work and watch these dogs die when you have opened either one or both sides of the thorax and then see how easily they can be revived by taking advantage of certain well-recognized principles, you will not have the confidence and skill in the management of your patients which you otherwise would and should have if you are to be the safest man managing surgical cases in your neighborhood.

Next you may work in the abdomen, and, after you have mastered the management of hemorrhage, you should do experimental work on the intestines, stomach, kidney, pancreas, liver, and spleen, where there is very much to be done. You will find each of these fields as luxurious a grazing ground as an untouched alfalfa meadow. When you ask yourself what is still left to be done, remember that nothing has been done compared with what is still left to be done in this enormous realm. Every day men come to me and ask, "What can I do?" There is scarcely any field of surgical work in which there is not a tremendous amount to do which can lead to reputation and to personal and professional advancement. Such work increases the good you can do as doctors in the community in which you live, and adds to the good reputation of your alma mater and of the teachers who instructed you.

Pardon me for leaving clinical subjects today in order to talk about these important matters in response to the kind words which I so very much appreciate, spoken by your president. If some of you will just practise these few suggestions I have outlined I shall feel amply rewarded for the time I have devoted to these topics today. I wish you godspeed in your hard but fruitful labor.

DIFFERENTIAL DIAGNOSIS BETWEEN BENIGN AND
MALIGNANT BREAST TUMORS BEFORE AND
AT OPERATION. VARIETIES. ATYPICAL FORMS.
CLINICAL COURSE. RELATION OF TRAUMA TO
CARCINOMA AND SARCOMA. SCIRRHUS

HISTORY

THE patient is a married woman, aged fifty-six. Her family and previous history are negative. In 1904 (ten years ago) the patient was thrown from a street-car and fell on her left side. She could find no bruises whatever, but could not sleep that night on her left side on account of "nervousness." In May of the following year, 1905, while bathing, she noticed a slight drawing-in of the breast just below the nipple, but could feel no tumor. In October, 1905, she noticed a slight mass, the size of a small marble, situated on the outer margin of the retracted area. Two months later, in December, 1905, she had pain and swelling in the breast and pus discharged from the nipple and at the site of the tumor. This discharge entirely cleared up in two weeks, but the tumor remained. Then she noticed that the whole nipple was retracted.

The tumor has gradually increased in size ever since, until it is now about the size of a goose-egg, hard, attached to the deeper tissues, not adherent to the skin, but distinctly adherent to the nipple. The nipple is covered with a scaly desquamation. The tumor occupies the position just back of the nipple. The breast is often painful. Below the nipple there is an area of retraction of the skin one inch long. The axillary glands are not enlarged.

She consulted a doctor two years ago, who said the retraction of the nipple was probably "reflex."

COMMENTS AND OPERATION

DR. MURPHY: How much swelling did she have? What do you mean by a *large quantity* of pus (the discharge from the breast in December, 1905)?

INTERN: It discharged for two weeks—quite a little every day.

DR. MURPHY: What do you mean by "quite a little"? Where did it discharge?

INTERN: At the side of the breast—in the upper quadrant of the breast.

DR. MURPHY: Did the discharge come from the nipple or did it come from the perforation of the breast? You say, "the side of the breast." That does not mean anything to me.

INTERN: She does not know.

DR. MURPHY: You ought to find out whether the patient had an open sore or whether the pus came out of the breast as milk would come out—through the nipple. That is an important differential matter.

That history is a very exceptional history for a mammary carcinoma, inasmuch as a period of ten years has elapsed since the beginning of the breast complaints. That is an exceptionally long period of time. There were, in connection with the development of the disease, however, two significant factors: One was a trauma without suppuration, and the other, thirteen months later, a suppuration without trauma. The initial trauma was not more than moderately severe, since it did not abrade the breast. As a rule, a contusion must be very severe to abrade the breast. She is fifty-six years old now. She was forty-seven at the time of the injury. She was past the menopause before this accident occurred—well out of the lactation period—and yet she had an abscess form slowly and finally rupture spontaneously. Then it healed, apparently, and the breast remained quiescent thereafter for a number of years. Two years ago a little enlargement of the breast was noticed. Since that time the breast has been slowly increasing to its present size.

This history is exceptional in that a long period of time elapsed between the original injury and the manifestations of carcinoma. It is exceptional in that suppuration came and went and the breast returned to normal size and remained quiescent for a long period of time. It is not exceptional in the fact that trauma is associated with its genesis. The type of trauma which precedes carcinoma of the breast is not the same type which precedes cancer in other positions in the body. After reviewing the etiologic factors of cancer, one comes to the conclusion that cancer usually is the sequence of repeated mild irritations, chemical or physical. That is the course of development of cancer in most locations. The rôle of trauma in the genesis of this disease in the breast, however, is a peculiar one. It seems to bear much the same etiologic relation to cancer of the breast that it does to sarcoma in many locations in the body. In cancer of the lip, tongue, stomach, skin, etc., there is usually a history of long-continued irritation, of frequent mild traumas, and, as a result of these recurring slight insults, either a displacement of epithelial cells through the basement-membrane takes place, or the basement-membrane is thereby so weakened that it becomes permeable to the growth of epithelial cells, and that constitutes the microscopic picture of carcinoma. With cancer of the breast, on the other hand, one finds the most frequent etiologic factor to be a single trauma of moderate severity, not mild, and yet not severe enough, usually, to produce a laceration of the skin.

We have rarely, if ever, seen a carcinoma arising in a breast where a previous operation was performed for a non-malignant cyst; nor have we seen cancer springing from a scar in the breast or from a stab wound; but, rather, it is an injury to the breast of the contusion type, producing a stinging or aching for a short time and leaving no immediate apparent disturbance of the gland, which finally, one and a half or two years later, at the point where the trauma occurred, is followed by the development of a carcinoma.

That relation to a single injury is true of breast cancer, but not of cancer of the stomach or uterus. It is a very frequent occurrence, however, in the history of all varieties of sarcoma.

You rarely have a sarcoma the sequence of mild, repeated irritations. A sarcoma follows a moderately severe trauma, not sufficient to produce a laceration, or, if it be a sarcoma of the bone, not severe enough to produce a fracture. One practically never sees a sarcoma develop at the site of a fracture. One often sees fractures occur at the site of sarcoma, but never the reverse. Therefore, it is an important aid in diagnosis to know from the history of the case the relation of the tumor to previous trauma. The trauma which precedes a bone sarcoma is usually of sufficient severity to give the patient a sudden shock, such that he will remember it later, as, for instance, knocking the leg against a chair. The shock passes off after a short time and the patient feels no further ill effect until, after a few months, a sarcoma begins to develop. Exactly the same sequence occurs in cancer of the breast.

The intern stated in the history that the tumor is fixed to the deeper tissues; but I find it not fixed to the chest-wall at all. There is an apparent enlargement of the gland due to a firm mass which I feel in it around and below the nipple. We shall send it to the pathologist at once to get a microscopic report. This mass has been increasing in size recently. There is present here the typical retraction of the nipple, so characteristic of breast carcinoma. We shall not make the usual incision. You see the drawing-in of the breast is of a rather peculiar type—it is transverse in location. Therefore, I shall make the incision a transverse oval with an upward extension along the outer border of the pectoralis major in place of the common incision, because this transverse incision will allow the skin-flaps subsequently to fit together with the least possible tension.

[Incision.]

[The usual method of removal of the breast and axillary glands and fat, as described in the February number of the CLINICS, was then performed.—ED.]

I always like to see the gross specimen and make my own interpretation of its appearance, and then contrast that with the interpretation of the pathologist. We both err, but from my study of the gross specimen and the clinical history I manage



Fig. 148.—The skin incision.

Figs. 148-152.—Radical operation for carcinoma of the breast. A series of pictures of Dr. Murphy's operation, including the use of pectoralis major and minor muscles to fill the axillary defect and prevent postoperative edema and neuralgia of the arm.



Fig. 149.—The lymphatic-bearing aponeurosis of the pectoralis major and the fascia and fat between the pectoralis major and minor have been dissected away, together with the breast, the surrounding fat, and the overlying skin. The costal and sternal attachments of the pectoral muscles are then divided with the scissors.

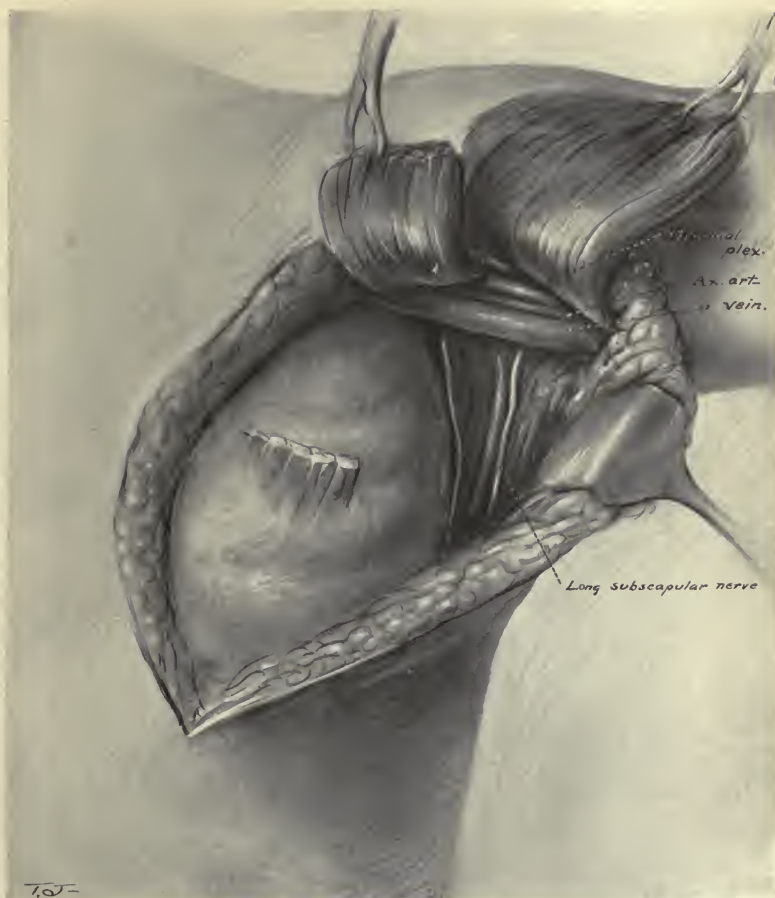


Fig. 150.—The pectoralis major and minor are dissected free from the chest-wall and clavicle and retracted upward and outward out of the field. The axilla then lies exposed clear up to the clavicle. The axillary glands and fat are then dissected *en masse* from the axillary vessels and nerves, beginning at the apex of the axilla and proceeding downward and backward. The long subscapular nerve is carefully preserved.



Fig. 151.—When the axilla is clean, the pectoralis minor is turned into the axillary space and sutured over the axillary structures by tacking stitches.



Fig. 152.—The pectoralis major muscle is turned in over the pectoralis minor and likewise tacked in place. A stab wound is then made through the lower skin-flap for tube-drainage and the incision closed.

to break about even. When the clinical history does not correspond with the microscopist's findings, then we usually side with the clinical history. Our observations have covered a number of years, so we have fairly positive convictions about it, based on the subsequent history of the cases. We always want the microscopist's report and have the greatest respect for it, but it is only one of the elements in making a differential diagnosis, because we have many tumors which, while microscopically malignant, are clinically benign, and vice versa. The correct solution finally works out. Tumors are reported again and again as non-malignant and yet finally terminate the life of the patient by recurrences, showing they were malignant. That is how we have come around to this conviction.

When the patient begins to vomit, as they often do in the first twenty-four hours, you will have some oozing, especially from small branches of the axillary vein. For that reason we always use a drain in these cases, introduced usually through a stab at the lowest angle of the wound. There is a mistake in that incision. As I swung around the tip of the cut I beveled the skin. That beveled portion will become necrotic, and it will be two weeks before it will drop off. Therefore, I just cut that off at the angle with the scissors. Beveling the skin is the most common cause of keloids. They are due more to the surgeon than to the skin. In the colored race you may have them very large, but not only in the colored race, but also often in those who have been nursed by a colored "mammy." That is a peculiar thing. One of the most severe cases we ever had of keloid was a young girl from Little Rock, Arkansas. When I looked her over and saw these great keloid masses I asked, "Who nursed you?" She said, "A colored mammy." That told the story. Why it does it we do not know, but it does it.

Let me go back again to where we started—the etiology of malignant disease. We have put on the board a classification of tumors of the breast. In the age incidence you will note that we have 11 per cent. of the cases occurring between the ages of twenty and thirty. I put that on the board to call your attention to a matter which is not sufficiently recognized, namely, the

frequent early occurrence of carcinoma. When you see that you have 11 per cent., or one-ninth of the total number, occurring in the third decade, it means that when you see a tumor in the breast you must plan to arrive at a positive diagnosis as to what it is, regardless of the age of the patient, and to arrive promptly—not to say, “Wait, we will see what it will be,” because if it is a malignancy and you wait, the mischief will be done before your diagnosis is made. If you are going to conserve the patient’s life, you must make the diagnosis of the tumor when you get the case. The time is coming when our sins of omission are going to be punished as severely as our sins of commission, if not more so, and I think justly more so.

In making the differential diagnosis of breast carcinoma there are many things you can avail yourself of to aid you. First, a clean-cut history of trauma is important in practically all the breast carcinomata. In those which occur in young women, married or single, you will find a definite history of a trauma in the early days, and a helpful point about it in that class of cases is that the trauma stands in closer relation to the final manifestations of the disease than it does in the aged, because it takes the aged longer to produce the gross manifestations after trauma than it does the young individual. I am speaking of the average case. When you find a tumor of the breast, that tumor must be diagnosed. (See the classification of tumors of the breast in the CLINICS for February, 1914, p. 181.) If necessary for the purpose of diagnosis, take it out. If, after taking it out, you have a smooth tumor, as hard as wood,—indeed, it is usually positively ligneous in its sensation to the hand,—that tumor is called, ordinarily, by the surgeon an adenoma. But an adenoma is a relatively rare disease of the breast. That hard round tumor in at least eighty times out of a hundred, perhaps ninety, is a cyst. If it is a cyst, all you have to do to make the diagnosis is to use an ordinary hypodermic outfit and puncture the cyst so its contents fill the syringe. That clears up the diagnosis. The cyst is primarily innocent; but if you will follow up the malignant tumors as indicated here in the later list, you will note that a cyst is not always innocent. You may find within the cyst a

papilloma, and that papilloma may become malignant; but in that case there is additional available information which you can get with the hypodermic. That fluid, when you draw it out with your hypodermic needle, is a grumous *bloody* fluid. Thus, usually, when you have a malignant papilloma lying within a cyst-wall, you get this additional information as to what is going on within the breast by the color of the aspirated fluid. Then the breast should be removed at the earliest convenient time for the patient, and all the wall of the cyst should be removed. Remember, I said in discussing the treatment of the breast that an incision in the breast does not lead to carcinoma. Do not bruise the breast. Pick up the cyst-wall and dissect it out. This kind of a trauma to the breast rarely, if ever, leads to malignancy. If the tumor is an adenoma, then you must take out all the capsule of the adenoma. Remember the relation of adenoma to adenocarcinoma, and that many of these types of adenoma border on malignancy. They may be microscopically malignant and clinically benign or they may be clinically malignant and microscopically benign. Either the pathologist or clinician may be in error. In order to be on the safe side you must take out all the capsule of the adenoma when you remove it.

There is another tumor which is often mistaken for an adenoma without careful microscopic examination, and that is sarcoma. A sarcoma is often encapsulated. It spreads, and, as it does, it forms a connective-tissue capsule on the outside. That is a most dangerous lesion and the most malignant lesion which occurs in the breast. You take out all the breast and you take out all the neighboring tissues; you clean out the axilla, and then the first thing you know the tumor recurs three, four, or six inches from where you took out the original tumor. In the management of all these tumors, particularly the sarcomatous type, the greatest care must be exercised not to compress the tumor mass at all until you have shut off the efferent lymphatics on every side of the breast, first on the axillary side and then from the other directions.

Let us agree that the diagnosis must be made promptly. No

procrastinating once you find the tumor—no excuse for procrastinating. The people consent readily to an operation. They are anxious to have it done. Therefore the whole responsibility is on the profession.

Let me mention one disease which is more outrageously managed than any other disease in the mammary gland, and that is the little red nipple which gets a little abraded and then a little more abraded, and two months, three months, six months later becomes a still greater abrasion. You first see a little crust. When you pick off the crust just near the outlet of the nipple, there is a tiny drop or two of blood, not so large as a pinhead. That area slowly increases in diameter, and finally it involves the circumference of the nipple. Then it spreads a little, covering the areola. It gets a little larger, and, finally, some one wakes up and tells the patient that she has a Paget's disease, when he should tell her that she has a Paget's cancer. Then she has salves, ointments, and applications, and she fools with it another six months, a year or two years, treating Paget's cancer as an eczema. That case is a cancer from the very first day that you see that little sore. It is the most wicked type of cancer which occurs in the breast. That little exudate which comes out and keeps coming is merely the discharge of the necrotic area which develops in that slowly advancing but terrifically malignant disease. Remember, Paget's cancer has a final cancer mortality of something over 90 per cent. as it is operated now. The patient, as a rule, first calls the doctor's attention to it, who treats it with pastes and salves, and it is not operated on until the axillary glands are involved and until the life of the patient is extremely hazarded. Now, if we as doctors know, and if we as doctors recognize, that Paget's disease is always a malignant disease from the very first day, then we should make the diagnosis and should remove the breast as soon as the lesion is discovered. Paget's disease should have a complete amputation of the breast the moment you see that secretion from the nipple, because that small scab is the beginning of a carcinoma, just as much as the crack on the lip or the chronic ulcer of the lip is the first stage of a carcinoma. That breast must all come out. If

the patient says no, then it is your duty to send her to some one else; but before you do so, make it painfully clear to her that it is a cancer and that the outcome will be fatal if she permits it to remain. You are then fulfilling your obligations. If you are positive enough, the patient does as you require. If you waver,—and patients have an instinct which tells them when you are wavering,—they do not do as you wish.

[A picture of a typical case of Paget's cancer, drawn from life by our artist, will be reproduced in color in the August number of the CLINICS.—ED.]

The pathologist reports that this tumor is a scirrhous carcinoma. Here is the gross specimen. You do not need a microscope when you look at that. You can make the diagnosis without a microscope. See that little focus! It looks as though it had a center in it of small millet seeds.

Let the record show that all the breast was removed; that there were enlarged glands on the posterior wall of the axilla; that the stumps of the pectoral muscles were retained; that the connective tissue and gland-bearing tissue beneath the pectoralis major and minor was dissected out.

You do not often have the glands on the posterior wall of the axilla enlarged. It occurs only in the deeper carcinomata. They are located, as a rule, on the anterior surface of the vessels. This gland was behind the artery and vein on the posterior wall of the axilla.

[NOTE.—This wound healed by complete primary union. There were no post-operative complications and no swelling of the arm, in which she already had a useful range of motion when she left the hospital, two weeks after the operation.—ED.]

THE DIFFERENTIAL DIAGNOSIS OF GASTRIC AND
DUODENAL ULCER BY DR. MURPHY AND DR.
CHARLES L. MIX. VALUE OF THE VARIOUS DIAG-
NOSTIC METHODS AND THEIR FALLACIES

HISTORY

THE patient, a married man, aged thirty-five, entered the hospital April 2, 1914, with the following history:

Ever since the patient was fifteen years of age he has been subject to severe attacks of pain in the epigastric region. These pains are burning in character, and come on from two to four hours after eating, increasing with hunger and subsiding on the taking of food. The patient says his most severe attacks have occurred when his stomach was empty. He can control the attacks by going to bed and by dieting carefully for a few days. These attacks come on usually in the early spring and in the fall of the year. Between times he always is conscious of a heavy, bearing-down sensation in the stomach region, especially after meals. He does not remember ever vomiting or being nauseated. He never has passed blood in the stools. The stools are always brown in color.

This condition continued until two years ago—in February, 1912. One evening, then, the patient was shaking the stove at home when he suddenly became terribly weak and collapsed on the floor. He does not know whether consciousness was lost or not, but is sure he had no pain. He was put to bed. The next morning he arose feeling very well, except for slight weakness. He ate a hearty breakfast and went to work. People noticed for a few days that he was very pale, and he remembers his heart pounding hard all day. On the second day he was too weak to walk, and went to bed and stayed there two weeks before he felt able to work again. On the second day after his weak spell he passed, he thinks, about one pint of blood in the stools, which were tarry in color. Chemical tests showed this to be blood. His hemoglobin was 60 per cent. after the hemorrhage, and he ran a subnormal temperature for ten days. He again went to

work and has had no pain since that time, but has had a great deal of tenderness over the appendix, gall-bladder, and in the epigastrium.

The following year, April or March, 1913, he was taken with chills, which would last from ten to twenty minutes, followed by a temperature of 101° to 102° F. every morning about 2 A. M. This condition lasted for about two weeks. Doctors thought at first it was typhoid fever, and then later diagnosed it as tuberculosis of the intestine. The von Pirquet test was positive. The patient worked all the time he was having the chills and fever. Since then he has worked and felt in the best of health. Being a physician, he has often made chemical tests for blood in the stools and always found them negative since the above attack. X-ray examination after a bismuth meal showed nothing abnormal. He has never vomited, never felt nauseated, and never was jaundiced. He has never belched gas, and never has had any distinct point of tenderness in the abdomen. He never has pain in his back, but has considerable trouble always with gas in the intestines, relieved only by vigorous catharsis or enemata.

DR. MURPHY: Dr. Mix has gone over this case and I shall ask him to go into the details with you.

DR. MIX: The history is suggestive of some abdominal disturbance which began when the patient was quite young. He does not, although the history may seem to imply so, have a very keen remembrance of the first fifteen years of his health history. He does remember that while he was in the medical school he had a good deal of abdominal disturbance at times. He remembers that these disturbances came on in the spring and fall of the year, and were, for the most part, associated with trouble in the epigastrium. In describing to me the sensations which began at the age of fifteen in his epigastrium he said that the pain was of a burning character, but he is so vague in his memory of these early disturbances that it will hardly be safe to come to a conclusion as to whether at that early time there was any pyloric spasm from abdominal disease or from an ulcer of the stomach or duodenum. The pain which he then suffered

while he was a medical student was *at times* situated in the lower portion of the right side of the abdomen, and by him was ascribed to his appendix. On such occasions the pain did not come on at any definite time, but occurred at almost any portion of the twenty-four hours, irrespective of food. By discontinuing his meals and by going to bed he was usually able to stop these disturbances for quite a number of years. Being a physician, he had a fairly good idea of his own pathologic condition, and always supposed he was suffering from a chronic appendicitis. Chronic appendicitis, of course, could have caused all the symptoms of which he then complained, including the pyloric spasm, which I am rather inclined to believe he had at times.

In the last three or four years the patient's history has become very much more definite. The pain in the epigastrium seems to have been more severe of late. It seems to have been confined to the epigastrium, because he states he has never had any pain in the back. The pain has been burning in character, coming on two or three hours after meals, increasing as he became hungry and diminishing as he took food. He was very careful to state to me that his pain was worst when his stomach was empty. He said that he had, at the suggestion of a physician, washed out his stomach religiously quite a number of times, with the result that the stomach always felt worse after being washed.

He also made another rather important observation, that he could stop the pain by going to bed. This symptom, stated in another way, amounts to the assertion that on assuming the prone posture the pain disappeared. He also found that if he dieted for a few days the pain would disappear. These two facts are always suggestive of ulcer, whether of the stomach or of the duodenum.

Another very important observation which he made was that he was distinctly worse in the spring and fall of the year. Elsewhere in the CLINICS I have alluded to the fact that duodenal ulcer has spring and fall exacerbations, just as does rheumatism; I believe that when the fact becomes established that duodenal and gastric ulcers are the results of infection, that the

reason for their incidence in spring and fall will be obvious to every one.

Another important complaint in his anamnesis is that of gas. He was very much bothered by its accumulation, but he could relieve it with catharsis or enemata. This intestinal gas of which he complained was probably the result of the associated constipation, which is quite uniformly associated with cases like this.

An important negative point in his history is the absence of nausea and vomiting, and especially of the vomiting of blood or the passage of tarry stools. He has no memory of ever having vomited blood. This point is of considerable diagnostic value, as I shall subsequently call attention to. He passed tarry stools only once.

During the month of February, 1912, he had a severe hemorrhage on the night that he shook the stove. This hemorrhage undoubtedly took place during the exertion which he made at that time, for he immediately became weak and collapsed. There was no pain, and he woke up the next morning feeling weak, yet having a fairly good appetite. He says that he ate a good breakfast. His pallor and his pounding heart clearly pointed to a loss of blood. It is not strange that he passed a pint of blood on the following day and that his hemoglobin percentage fell to 60 and that he had a subnormal temperature for ten days. He rapidly recovered from this severe hemorrhage, and things went back pretty much to where they were before the hemorrhage, his pain in his epigastrium still continuing to give him trouble in the following fall.

Again in March, 1913, a new set of symptoms appeared. These consisted of chills lasting from ten to twenty minutes, with a temperature rising to 101° to 102° F. Strangely enough, these chills came at uniform intervals. Every morning at 2 A. M. his temperature rose to the highest point and subsequently fell to normal. For two weeks he had these chills. He states that typhoid fever was suspected, but finally the diagnosis of tuberculosis of the intestines was made, based largely on a positive von Pirquet. He, however, did not believe that he had tuber-

culosis of the intestines, because he was able to work all the time and felt fairly well. During this period he had no nausea or vomiting, he passed no blood, and was not jaundiced.

The interpretation of this history leads us to the conclusion that he originally suffered from an appendicitis. His appendicitis seems to have been of the chronic obliterative type, beginning when he was fifteen years of age, being associated from time to time with pyloric spasm. There seems never to have been an attack of very severe appendicitis with abdominal rigidity and vomiting, beginning as a generalized pain and localizing within eighteen to twenty-four hours in the appendiceal region. He seems rather to have had an appendix which was in a state of chronic irritation, with a gradual obliteration of its mucous membrane and lumen. I shall expect, when his abdomen is opened, to find that he has an atrophied appendix.

Physical examination at the present time in this patient's case shows tenderness over his appendix, also over his epigastrium and over the duodenal area. When he takes a deep breath and the gall-bladder area is tapped vigorously, pain is caused, indicating presumably either a periduodenitis or a pericholecystitis, or both. At the present time repeated examinations show no blood in the feces. The stomach analysis shows a marked hyperacidity, but no blood.

Returning again to the history, we find that previous to February, 1912, a set of symptoms developed in the patient which are almost pathognomonic of duodenal ulcer. These symptoms were severe pain in the epigastrium, coming on from two to four hours after meals, being increased by hunger and diminished by the taking of food, and worse when the stomach was empty; relieved when food was stopped for a few days; coming on mainly in the spring and fall of the year; associated with no nausea, vomiting, or jaundice, but accompanied by constipation, gas in the bowels, and belching. These are classic symptoms of duodenal ulcer. When to them there is added a distinct history of hemorrhage from the bowels, such as we know took place in February, 1912, there is no escape from the necessity of making a diagnosis of duodenal ulcer. In February,

1912, one might have been disposed to look upon the case as one of chronic obliterative appendicitis with a secondary infection of the duodenum and the production of a duodenal ulcer, the result of this ulcer being a very severe hemorrhage, followed by a fainting attack and the reduction of the hemoglobin to 60 per cent.

A new factor came into the case in March, 1913. This consisted of chills and fever, which were of the type usually associated with an infection of the ductus choledochus or of the neck of the gall-bladder. These chills definitely recurred for a period of about two weeks at exactly 2 o'clock every morning. It was not the type of fever that occurs in tuberculosis of the intestine, which diagnosis you will remember was made. In tuberculosis of the intestines the temperature usually reaches its maximum between 2 and 4 P. M. Furthermore, it is insidious in origin, and is not associated with chills definitely recurring at stated intervals.

What was the cause of these chills? Doubtless, following the ulcer of the duodenum, there was an extension of the infection, which resulted from the ulcer, up through the common duct. This infection may have spread in the submucous tissue of the common duct without the production of a jaundice, or it may have come by the lymphatics from the duodenum to the transverse fissure of the liver, into which the lymphatics pass. This infection was not explained at the time it took place; we have no data as to the physical findings at that time, and we should like much to know whether there was any gall-bladder tenderness on this occasion. Our experience is that these fevers with definite, recurring chills occurring at stated times, precisely as they take place in malaria, are due to a sudden infection of the blood-stream. You will notice the type is exactly the type that occurs in malaria, where there is a definite chill at a stated time every day or every second or every third day as the case may be. In the cases, like that of this patient, where the chill occurs every twenty-four hours at a stated hour and where there is no history of malaria, there is almost invariably to be found an infection either of the common duct, the neck of the gall-bladder, the

urethra, or a direct infection of the blood-stream from some focus in the body. The chill is exactly comparable to the chill that is obtained after the injection of too strong a vaccine. The reaction is doubtless almost as quick as the reaction which is obtained after the systemic injection of such a vaccine. Knowing definitely that the patient had a temperature at this time, furthermore knowing that gall-bladder infections and common-duct infections are extremely common as the sequence of duodenal ulcer, I have no hesitation whatever in saying that in March of 1913 this patient's common duct probably was infected. Evidently the infection cleared up, and presumably it has left behind it no cholecystitis and no gall-stones, because there is at the present time no palpable enlargement of the gall-bladder and no very marked tenderness over it. There is some tenderness in the right upper quadrant, such as is to be found in a pericholecystitis or periduodenitis, but there is no history of a gall-bladder colic or of severe gall-bladder pain; and at the present time there is no marked tenderness over the gall-bladder itself.

One observation was made during the run of this fever, which the history says nothing about, namely, the leukocyte count. During these two weeks of chills the leukocytes never ran above 9000 per c.mm. This fact is in favor of the diagnosis of infection of the common duct or of the neck of the gall-bladder, rather than opposed to such a diagnosis, because we find in these infections that the leukocyte count does not run very high.

The diagnosis of tuberculosis of the intestines based on a positive von Pirquet is not tenable. Most adult patients are likely to give a positive von Pirquet without having a particle of active tuberculosis about them.

The diagnosis seems to have been, then, a chronic appendicitis coming on when the patient was fifteen years of age, lasting for a number of years, and subsequently giving place, and possibly giving rise, to an ulcer of the duodenum, from which a hemorrhage took place in February, 1912. Secondary to the development of an infection of the duodenum there seems to have been an infection of the ductus choledochus or of the neck

of the gall-bladder, or possibly a cholecystitis. The probabilities are that the present pathology will show nothing more than some adhesions, probably a periduodenitis or possibly a pericholecystitis, which will account for these troubles of a year ago. There may be more than that. I do not think there is much pathology about the gall-bladder at the present time on account of the fact that there is no localized tenderness. There is some diffuse tenderness, however, in the right upper quadrant and this would strengthen one's belief in the presence of a periduodenitis rather than of pericholecystitis. We incline to the opinion that the appendix will have to be removed, the duodenum exposed, and probably a gastrojejunostomy will be advisable. The last point has to be determined at the time of the operation, of course.

[Incision. The abdomen is opened to the right of the mid-line, in the usual manner, and the stomach is brought out through the incision.]

DR. MURPHY: I am not personally familiar with this case from a clinical standpoint, but you do not need to know the case clinically to see this ulcer. There it is! See the roughened peritoneal surface over it! There is the depression accompanying it, just beyond the pylorus. See the stellate scar, just like a text-book picture! It has produced a stenosis, not a complete but almost a complete closure—look!—of the duodenum. That is beautiful!—beautiful to look at and beautiful in its close agreement with the clinical diagnosis. Let us see whether there is an additional ulcer farther down. Sometimes you have two or three of these duodenal ulcers; but most of the time you have only one. There is the rest of the duodenum coming into the field; no adhesions on the sides and no thickenings.

Let us examine the gall-bladder. Let the record show that the gall-bladder came readily into my fingers; that it is elongated, but readily compressible. When it is easily compressible, then you feel safe about the gall-bladder. When it is not readily compressible, you have to go over it again and again to exclude a lesion. There are no stones in this gall-bladder.

The next question is, is there an ulcer in the stomach? Here

is the point (on the lesser curvature near the pylorus) where ulcer occurs most commonly. There is no evidence here of adhesions. Let me get the under portion of the duodenum on the pancreatic side. There, occasionally, opposite the one ulcer, you may have another ulcer.

Let the record show that there is some thickening of the head of the pancreas; that this thickening is not woody at all; that it feels as though it were bunched rather than infiltrated. Nothing pathologic there.

There is the pyloric vein. The ulcer lies just outside it. There is the pancreas, you see, extending down to the inner side. This was an infected, hidden ulcer, producing stenosis. Because it does produce a stenosis, there is no question as to what should be done so far as the operative procedure is concerned. We must take steps to prevent the further retention of food in the stomach which would finally become complete. (The consequent operative technic—the oblong button gastro-enterostomy was practically identical with that already described in the CLINICS.)

The reason we have gone back to the button is that while we, like every one else, have been all over the field, both with the suture and with the button, we have come back to the oblong button in all gastro-enterostomies because of its low mortality. The greatest mortality in gastro-enterostomy today is from pneumonia. The cause of mortality second in frequency is hemorrhage into the bowel and stomach. We do not see how we can avoid these pneumonias. They occur with local anesthesia, they occur with ether, and they occur with spinal anesthesia. We believe they are due to an infection, namely, that they are embolic, entering the circulation from the local field. They occur much more frequently with laparotomies in the upper abdomen than in the lower abdomen. We are unable to prevent them by anything we know of now. We *are* able to avoid hemorrhage. By the careful application of the suture, according to recent methods, we have been getting good results, but not perfect results, against hemorrhage. We cannot have hemorrhage into the stomach as a sequence of the button operation.

While on the subject of the button it is *apropos* to state that Dr. Murphy received recently the following letter:

THE JOSEPH PRICE HOSPITAL,
J. W. Kennedy, M.D.,
241 N. Eighteenth Street, Phila.

JOHN B. MURPHY, M. D., Chicago, Ill.

My Dear Dr. Murphy: During my twelve years' association with the late Joseph Price I was constantly a witness to use of your button for anastomosis. I have no recollection of a single instance in which the button gave us any post-operative trouble. Are you saying enough about its use? As you know, Dr. Price was probably the greatest advocate of its use and used it hundreds of times. I feel that the button is altogether the safest method of anastomosis in any one's hands, and also the easiest method for those with meager experience.

I constantly advise its use to men who visit my clinic in the J. P. H. Many of these men are doing admirable work in small towns over our country, and it is necessary to place the most substantial methods in their hands.

Faithfully,

(Signed) J. W. KENNEDY.

Dr. Mix's interpretation of the case is perfectly right—correct in every detail. Duodenal ulcer is a very common disease, and not only a very common disease, but very commonly not recognized. I cannot tell that better than just to cite my experience from a little town in Iowa. A doctor out there has brought in five duodenal ulcer cases all correctly diagnosed from a little town of 3000. If he has five cases in a town of 3000, what is becoming of the 5000 or 6000 cases which are occurring in a great city like Chicago, where there is not one duodenal ulcer diagnosed for every 30,000 or 40,000 inhabitants? The reason they are not diagnosed is because we first called all these stomach diseases "dyspepsia," until that term became unscientific and unfashionable, then "gastralgia," and, as that also became unscientific, "hyperacidity," and then "gastric and duodenal neuroses." So commonly is that last term used, particularly in scientific Germany, that it is now considered synonymous with duodenal ulcer. Therefore, when you make a diagnosis of gastric or duodenal neurosis, it means, I believe, when interpreted, that your patient probably has a gastric or duodenal ulcer, just as when, ten or fifteen years ago, you made a diag-

nosis of gastralgia only to find out later that the patient had gall-stones.

I will ask Dr. Mix to have the history reread and to accentuate again the points in the diagnosis, because to me all of medicine centers around the translation of symptoms and signs into pathologic entities. That is the greatest achievement there is in medicine.

(History is reread.)

DR. MIX: The first statement in the history is at first sight misleading. Please understand that the first paragraph refers to the *present* complaint of the patient rather than a careful history from the time when he was fifteen years of age. The statement made in the history is that ever since the patient was fifteen years of age he has been subject to severe attacks of pain in the epigastric region of the abdomen. I have already explained that the early history of this patient's case is not that of severe pain in the epigastric region. Such pain did not come on until later in the course of his disease. The original complaint when he was fifteen was of a series of vague disturbances consisting mainly of burning pain coming on at any time, mostly in the spring and fall, and occurring in the epigastrium and associated at times with tenderness over the appendix. We concluded that all the early history belonged to the appendix as a chronic appendicitis. Later on a new set of symptoms appeared in the history, consisting of pain coming on at a definite time, being associated with irritation on the taking of food and aggravation on gastric lavage. This is the history of a duodenal ulcer, and we find the history confirmed by the appearance of a hemorrhage in February, 1912.

In the next stage, as this patient shows, we very frequently find an association of three things—a chronic appendicitis, a duodenal ulcer, and a gall-bladder infection. We do not ordinarily try to differentiate *between* these things, deciding as to which *one* of the three the patient has. Rather, we try to figure out what combination of these things the patient may show. It is usually not just an appendicitis or just a duodenal ulcer or just a gall-bladder, but some combination. Frequently the combination

is an appendicitis and ulcer, but still more frequently a duodenal ulcer and a gall-bladder. In this particular case all three seem to have been present. First, there was an appendicitis; second, a duodenal ulcer; third, an infection of the biliary tract. The duodenal history is perfectly clear, with the very characteristic hemorrhage which occurred.

One important point I think may be emphasized: there was no pain in the back in this man's case, because the ulcer was situated on the *anterior* surface of the stomach, away from the back. I think we shall find on careful examination of the histories that those cases with back pains are the ones in which the ulcer occurs near the head of the pancreas and more toward the posterior wall and away from the pylorus.

A third step in his case which took place last March was the development of an infection of the biliary tract. Why was that not a tuberculosis at that time? Because of its very stormy onset. Tuberculosis does not have such a stormy onset, with chills and fever. Why was it not a typhoid? Because the chills came on early. Typhoid chills occur late in the case, never early. Consequently neither of these maladies could be suspected and they can be ruled out. Malaria could be suspected, for, in the patient's case, the chills occurred in March, at a time and place where malaria does not occur. Had he lived in the South, some physicians would inevitably have made a diagnosis of malaria. The possible cause for that high temperature with chills, the chills being periodic, is very apt to be, in the absence of a malaria, an infection of the biliary tract or of the urethra or of the pelvis of the kidney or some focus where an immediate infection of the blood-stream can take place, giving a reaction quite as pronounced as that which is obtained with a hypodermic injection of a vaccine. It is the same general principle. We did not expect to find any marked infection of the biliary tract, because of the absence of any biliary tract symptomatology at this time, but at the time he had the chills there was some such infection. There was no blockade of the common duct, because there was no jaundice. There was no production of stone, because there was no passage of stone and because no stone was

found just now. The infection, as I said before, was associated with the comparatively low leukocyte count of 9000, which we regard as of some value in these cases. We do not find, particularly in these infections of the biliary tract of this type, a very high leukocyte count.

To recapitulate, as I did before in speaking of my observation of the case, it seems that there was primarily his old appendix trouble, which he had early; that this was followed by a duodenal ulcer; and, as a third etiologic factor, he developed a biliary infection, which healed subsequently. Had the duodenal ulcer not now been operated upon, it would have gone on and on and he would undoubtedly have developed a common-duct infection. We did not take an *x*-ray, because he told us that repeated *x*-ray pictures were negative. That may be true.

Dr. Murphy, in his operation, noted that the pylorus was rather stenosed. There was, however, no apparent dilatation of the stomach and no regurgitation or vomiting of food, or other reason for suspecting the existence of a pyloric stenosis other than the pyloric spasm which was present. The pyloric spasm itself, perhaps, is to be looked upon as one of the consequences of the narrowing pylorus.

DR. MURPHY: That covers the diagnostic phase of this case very thoroughly. The findings here at the operation fitted in so nicely with the symptomatology that they make this a very gratifying case. When the diagnosis is correctly made, it is wonderful how the pathologic findings confirm the clinical history; and when they do not, there is something wrong in Denmark. Some of you gentlemen were here on Monday and recall that patient who came in as an appendicitis case or as a cross between an appendicitis and a gall-bladder case, and you will remember that the patient was here ready for operation and we did not operate. We did not operate because the clinical history did not fit the diagnosis made of the lesion. The clinical history did not fit the diagnosis because the patient had pain on the right side. The patient had severe pain on the right side. She had nausea and vomiting, and sensitiveness to pressure all over the right side, clear down into the pelvis, and along the zone where the appen-

dix should be situated, The patient had had a temperature of 102° F. in the course of the attack, and when she was up here I think it was something like 101° F., with a leukocyte count up to 16,000. You may say that course of events looked like the clinical history of an appendicitis. No, it did not. Why? Because the patient had had pain on urination—frequent urination and an evident cystitis—for something like two weeks preceding these abdominal symptoms; but since the pain began in her side high up, the pain in the bladder had diminished and had apparently become a matter of lesser significance. Yes, lesser, but only by comparison. We hesitated about the diagnosis. We had a urinalysis made and it showed from 12 to 15 pus-corpuscles in a one-sixth field. We said we could not differentiate that case from a kidney lesion, and if we had to diagnose it at all we should call it a lesion of the kidney. It was an ascending infection from an infected bladder. The next morning we made a cystoscopic examination and there was a plug of pus over half an inch long protruding from the right ureter, showing how important it is to get the whole story of the patient's illness before we arrive at a diagnosis.

That case was similar to another patient of ours, who gave us a trying experience which helps me feel convinced that the diagnosis must be made on the clinical history, as well as on the physical findings. I was called up at 6 o'clock one Sunday morning to operate a case of appendicitis. Now, 6 on Sunday morning is not a nice time to be called up, even for an emergency case of appendicitis. But anyhow I went, and this was his story: He had had several attacks, and the previous summer, when he was driving along the Hudson in his automobile, he had an attack in the car between Albany and Troy. The attack was so severe that he got out of his car and went into a farmhouse and got the housewife to put some hot stovelids and hot plates on his abdomen. Then, in an hour, his pain subsided and he resumed his journey. That history is all that saved me from opening his abdomen. If appendiceal pain is severe enough to double a man up and to make him go to bed, it does not stop in half an hour. It continues and he remains sick for days, not hours.

He had another attack after he came home, less typical in the clinical history. We examined him and over the normal location of the appendix there was great sensitiveness. That morning his temperature was 100.5° F., and his leukocyte count 11,000; but when we sat him up on the side of the bed with his legs hanging down and made fist percussion over the kidney, he gave the typical response for a kidney lesion. Then his urine was examined and found full of red blood-corpuscles. The x-ray did not show a stone, but on the third day he passed the stone from the urethra. It was a uric-acid stone. I merely cite these two cases to call your attention to them and to show how easy it would be to err and to go in and take out an appendix if we had not analyzed the clinical history. It is analyzing the clinical history which saves one from many errors where the symptoms appear to be typical of an appendicitis. It is analyzing the clinical history which saves one from having the patients come back with the same symptoms, after you have removed the appendix, which they had before the operation, and causes the patients not unnaturally to look a little askance at the doctor. And yet they are rather generous. They admit we may err and are generous even when we do; and we err chiefly by failing to do in a systematic way the little things which would enable us to arrive at a diagnosis.

Here I have put on the board a chart of the different symptoms which occur in connection with duodenal and gastric ulcer. (See April CLINICS, pp. 270, 271, 272.) I have arranged them here so that you get them in a definite way. You will see that pain is the most common of all the symptoms. Pain is present in 94 per cent. of gastric and duodenal ulcers. You will see that emesis is present in only about one-half the cases. Then you will find that hematemesis in both types of ulcer occurs only in about one-fifth of the patients. Then you come to the presence of blood in the stools—melena. Melena means the recognition of blood as gross blood in the stool, which Dr. Mix spoke of this morning in connection with this patient who had a severe hemorrhage. That is about what you can obtain in the way of information from these patients. When you come to the presence

of occult blood, I think 81 per cent. is low, particularly if you are including all your duodenal cases. It is the occult blood which gives the best information. When you come to the chemical findings, to the acidity which is present, the hyperacidity and the subacidity, you are able to get but very little information from the laboratory. The laboratory has been a disappointment to us in the differential diagnosis of ulcer in every point except the presence of blood. In other words, the diagnosis has to be made chiefly on the clinical symptoms and history. You can see that this small percentage of hyperacidity and subacidity just about balance each other, so they add little or nothing to the positive information one receives from the laboratory tests. You remember at one time we thought that the laboratory tests were going to be of great value.

Now we come to another method, of which we have not the statistics, and that is the Rieder bismuth meal with fluoroscopic examination. The bismuth test is of comparatively small value in many of the cases in which we most need help—that is, the early cases. In the cases of ulcer of the stomach producing obstruction, yes. In the deep carcinomata of the stomach, yes. In the perforating ulcer which burrows from the under surface of the duodenum into the pancreas, yes; but these are all late pathologic conditions. If one waits to make a diagnosis until the time when these things show, he is able to do often little or nothing for the patient. Therefore, we must come back again, after we have made the fluoroscopic examination and the *x*-ray plates and all that, to the patient's story and analyze the relations of the different symptoms, especially their relations to the intake of food and the effects of food.

That chart which you see on the board is discouraging; but if it represents the facts as we believe it does, then let us accept it and then let us remember that we must analyze these symptoms in the order in which they occur, and the severity of the disturbance of the stomach function when there appear but few physical signs. Where you have an obstruction of any considerable degree, you not infrequently have a dilatation of the stomach. That is easily made out with the Rieder bismuth meal and just

about as easily with a seidlitz powder, in fact I have as much confidence in a personal administration and observation of the obstruction test by a seidlitz powder as by bismuth. The patient who has a duodenal ulcer with symptoms of an obstruction holds the gas in his stomach and has pain and often severe pain before the gas escapes. We find in interpreting these gas symptoms that they have a very pronounced relationship to duodenal ulcer. They also have a pronounced relationship to gastric ulcer when the ulcers are of good size. Such patients get very severe pain when you put in the double seidlitz powder. In making that test, therefore, one must always make it with care. I never do it without a little creepy feeling that some day the stomach is going to burst when I am making the test, but I take this precaution. When I am ready to give the seidlitz powder, I always have one of my associates standing on the opposite side of the table with a basin and a stomach-tube. He is standing there with no other purpose than to put the tube in the esophagus when the patient complains of severe pain. You practically never go into the stomach with the tube: just touch the posterior wall of the pharynx. The moment the patient expels the gas, the pain is relieved. Even then sometimes you have them collapse and turn white. Sometimes it is half an hour before you can take them off the examining table. That is the case with acute ulcer. We have had two cases of bleeding in these many years, but never had any serious accident. We have never had a perforation, but we always have a fear of perforation.

[The wound healed by primary union. The patient left the hospital April 23, 1914, completely relieved of his previous symptoms. The acid content of his stomach was still a little high, however, but he could eat full meals of general diet without any discomfort and was a regular attendant at the clinics from the time he got out of bed until he left the hospital. The button was passed after he left the hospital, thirty-one days after it was put in.—ED.]

THE DIAGNOSIS OF PREGNANCY IN A TUBE OR A
BICORNATE UTERUS ASSOCIATED WITH FIBROID.
THE DIAGNOSIS OF THE COMPLICATIONS AND
THE MANAGEMENT OF THE CASES. ABORTIONS.
SLOUGHING OF INTRA-UTERINE FIBROID. ENU-
CLEATION. CONSERVATION OF UTERUS

HISTORY

PATIENT, female, aged thirty-three, married, entered the hospital April 3, 1914.

Menstrual history began at fifteen; every thirty days, regular, from five to seven days' duration, three napkins a day, always painful. For it she was operated in October, 1913. The findings, as reported at the operation, were a double vagina, which was made into one, a possible bicornate uterus, and a fibroid. A curetage was performed. After this operation she had no pain during the next period, November, 1913, and she flowed only two days, using one napkin a day. In December and January the periods were normal in amount and duration.

During the first two weeks of February, 1914, for one-half hour after rising in the morning, she felt slight, cramp-like pains in the lower abdomen, which resembled the cramps she had during menstruation. She did not menstruate during February. She remained perfectly well until the middle of March. She never was nauseated, nor did she vomit during this time. When the menses did not appear after two weeks of March had passed, she consulted a doctor, who examined her and said she was pregnant. He examined the breasts, and then for the first time she noticed that they were enlarged and that the nipples were darker than usual; but there was no secretion in the breasts. On March 24th she noticed a small lump on the right side of the median line of the abdomen, just above the symphysis pubis, which was hard and about the size of a walnut. On Sunday, March 29th, shortly after rising, she began to flow. The flow continued all day, and she had cramp-like pains over the whole of the abdomen. She used seven or eight napkins that day. She went to bed about noon and remained there all day. That

night, about 10.30, she passed, after considerable pain, a couple of large clots, after which she felt relieved. A doctor was called, who said he found decidual tissue but no fetus in the clots. She continued to flow from that time, with slight cramps, until Wednesday, April 1, 1914. On that day she was taken suddenly with violent stabbing pains. She would be perfectly quiet for a time, and then suddenly she would start up, saying she had one of those stabbing pains. These pains continued until Saturday morning, April 4, 1914, when they stopped. She flowed a little on the Thursday and Friday preceding, but has not noticed any blood since Saturday, April 4th.

The abdomen is slightly enlarged. A hard mass can be felt in the midline just above the symphysis pubis. On each side there are small masses, also hard, about the size of an English walnut. She has some distention from gas in the upper abdomen.

COMMENTS

DR. MURPHY: Let me just consider this case more in detail. A woman of thirty-three, married about one and a half years, was examined by a doctor last August who found she had a double vagina. The septum was divided, but it was not determined whether she had a double uterus or not. She had a fibroid present at that time. The case went along until the seventh day of January of this year. She menstruated regularly, the full amount in the normal way. She skipped the February menstruation. She skipped the March menstruation. You see it is not quite up to her April time, so it is not quite three months. I think it was on Monday of this week she began to flow a little. She flowed a little more, and then she had some pain and then she expelled some material which the doctor pronounced decidua. Then she began to have stabbing pains in the right side of her pelvis, very peculiar. I saw her have some of them last night. She would be perfectly quiet and then suddenly she would jump, because of the sudden stabbing pains. She is still flowing a little.

On examination a fibroid is felt which fills the basin of the pelvis and is also felt as a mass coming a little above the

symphysis in the midline. On both sides in each inguinal region are hard nodules which feel as though they lie under the skin of the abdomen, though I believe they are inside of the abdominal wall; but they feel almost as if protruding through the skin. The doctor examined her some six weeks ago, and then the fibroid could not be felt, as I believe, above the symphysis. Now it comes well above,—two inches, I should say,—with the hard nodules. Examination by the vagina reveals only one palpable cervix. Whether there are two cervixes and whether it is double uterus I do not know, but the fact that she expelled the decidua is suspicious of the presence of a second uterine cavity, so that although she may be impregnated in the tube, she may also be pregnant in a second cornu. On account of the fibroid enlargement which has taken place the uterus now extends up to the right rather than to the left. It looks as though she was impregnated either in a tube or in one cornu of a bicornate uterus, and that the decidua came out from the other cornu. Then, to complicate matters, there is a fibroid which blocks the pelvic outlet. You can feel nothing but a hard, solid wall filling all the pelvis—woody hard, and nowhere soft. The little stump of the cervix which seems to come out from that wall is solid. It is believed that she has been pregnant on account of the missing of the menstruations. We have sent her blood to the Columbus Laboratory to have the Abderhalden test made. Here is one of the places where it is really valuable to know the result of this test.

If the test shows that she is or has been pregnant, the question is what to do. If she has a double uterus, she may go on to gestation and to the completion of gestation with the fetus in one-half of that uterus, which would be very desirable. She is thirty-three, and this is her first pregnancy. If an operation is done now, it must be an operation for the removal of the whole pregnancy and uterus and she will never have a child. That she cannot have a child by the normal route is clear, because the outlet to the pelvis is completely blocked with the fibroid. If she is pregnant, the question is whether to operate now and remove the uterus with the fetus, or to let her go on and keep

her under observation in the hospital, and keep in fire-alarm readiness, waiting to go in and open that abdomen at any time. I believe that the fetus and uterus can be taken out at the completion of gestation as safely as it can be done now. You have the danger of rupture in the mean time. Removal of the fibroid is not easy. If she has an impending miscarriage, which is causing the flowing, or if flowing or not, the case is serious.

As to the management of this case from now on: The examination was made last night after carefully cleansing the hands, putting on gloves, and preparing just as one would prepare to do an abdominal section. After all that was done, and from now on, she is to have a sterile dressing on the vulva. After each urination the vulva will be washed off with lysol. Why? Because if she has an impending miscarriage and she gets an infection from repeated examinations, she will lose her life in all probability. That is one of the great dangers of a miscarriage. Remember that in 1882 Schroeder,—the professor of gynecology in Berlin,—the greatest teacher I have ever listened to except one, Professor Haynes, of this city, professor of chemistry at Rush—was teaching the proper management of miscarriages, and I must say that I think he used the most sane methods and recognized most clearly the important factors in the general preparation and management of these cases that I have heard of from that time until now. In speaking of miscarriages and their treatment he said: "When the patient has an impending miscarriage without other pelvic conditions which force you to make an examination, no examination of the condition of that vagina or uterus must be made." Never put your hands across that threshold, because you will carry into that vagina, in doing so under ordinary circumstances, saprophytes. Decomposition takes place and then, following the miscarriage, comes infection. The infection of a miscarriage has its source from without. If the miscarriage is going to take place, your examination will do no good. If it is not going to completion, your examination can do harm. Keep still and let the patient alone. When you do examine a patient with a miscarriage and some of the products of conception are

retained, then do not stop until that uterus is cleaned out and completely cleaned out. Now you can see the reason for it; because if the work is only partially done and remains of the fetus and placenta are left in, the patient may get a sepsis which will cost her her life. Remember the fetus may be dead in the uterus for weeks and months and may be dead in the abdomen for years, and never do the slightest harm to the patient so far as sepsis is concerned, because sepsis is a matter of admission of microorganisms from without. I think that point ought to be printed in the Ten Commandments which the doctor receives when he starts to practise medicine, and which he should read every day before breakfast if he is going to do active practice, because the temptation is so great, when you see a patient with an impending miscarriage, to examine the condition. Let the patient alone absolutely if you are going to keep the infectious microorganisms out of the field.

It is an entirely different thing if the patient has attempted an abortion with instrumentation. These instruments are usually as filthy physically as the person who uses them is morally. Then your cue is to go in and clean it all out, which is an entirely different situation.

I have suffered for following these rules on a number of occasions. I remember once on Morgan Street I was called to see a woman who had an impending miscarriage. I did all the washing off of the vulva and put antiseptic dressings over the vulva and went home. The next morning when I came the woman's husband met me at the door and said: "We do not want you. We got a real doctor last night, who came in and examined the patient." I think it was the fifth day afterward, when I was passing I saw there was crape on the door. He examined the patient. I merely cite that case to accentuate the point I made about examinations. Keep your hands out.

That is to be the plan in the management of this case until we determine whether we have a pregnancy, which will take us until tomorrow night or early Monday morning to find out. It takes that long to make the Abderhalden test, which is a very important thing to supply in such a case. Then we shall deter-

mine whether she is going to have a miscarriage or already has had one. If she is not, we shall tide her over without examination. If she has a miscarriage, the placenta will come away. Then we shall wait until she is fully over that event before we do a laparotomy. Then we shall have the proposition before us of whether to do a myomectomy or a hysterectomy. But if you go in while the fetus is still there, you have no choice—it must be a hysterectomy.

To accentuate the dangers of doing this operation in the presence of a pregnancy with a possible sepsis present you have to think only of the mortality which occurs in doing an operation for an acute appendicitis in the presence of a pregnancy. You will remember that some two years ago we took up the consideration of this subject in our volume of the Year Book of Practical Medicine—Appendicitis in Pregnancy. The mortality is something frightful. It is frightful when you operate and when you do not operate. It is, on the whole, more serious without an operation. Now, here is one of the reasons why the mortality becomes so great. If you are going to operate at all, you must operate in the first few hours of the appendicitis. You must take a positive stand for or against the operation in the very early hours, because if you wait until the pus is outside the appendix, you have a 40 per cent. chance of losing your patient. Think of it. It makes one shudder to think of a 40 per cent. chance of losing a surgical patient. If you make the diagnosis early and decide the patient is to be operated, then do the operation before the pus has escaped beyond the boundary of the appendix. The patient will probably get a miscarriage. Many of them do; some do not. When they have a miscarriage, the microorganisms circulating in the blood lodge within the uterus, and you have a septic process in the soft, dilated uterus, a thrombophlebitis, an escape of the infected infarcts into the lungs, and the patient dies.

I merely bring out these points in diagnosis and management because they fit right in with some of the phases of this case which is in the house now waiting for developments. Everything is prepared so that if the cornu of that uterus does rupture,

we shall be ready to care for it, just as in a tubal pregnancy, a case of which was described in the December number of the CLINICS. We described another case, giving the Abderhalden test in detail, in the April number of the CLINICS.

[The case progressed for over three weeks without unpleasant symptoms other than the occasional stabbing pain in the lower abdomen, for which small doses of heroin had to be administered, and a slightly bloody show at intervals of from two to three days.

On April 25th a digital examination was made and some shreds of material were found protruding from the uterus. On April 28th, three days later, the patient had an elevation of temperature to 101° F., an odor to the discharge, more flow, and more severe pain in the lower abdomen.

An examination was made, and it was found that a sloughing fibroid was being expelled from the cervix of the uterus. After a careful preparation of the field and of the vagina, the fibroid was enucleated by digital manipulation and traction with the Nélaton forceps. It was attached just inside the internal os. There was but little bleeding following the enucleation. The vagina was thoroughly sterilized with lysol douches, and these were continued for the next three days; by this time the odor had entirely disappeared from the discharge. The temperature, which had reached 102° F. on the evening following the operation, dropped to normal on the third day and showed no subsequent rise during the remainder of the patient's uneventful convalescence.

There are still a few small fibroids palpable on the surface of the uterus, but otherwise the uterus appears normal.

This is a fortunate outcome for this patient, since the uterus is conserved and the woman's ability to have children has been maintained.—ED.]

THE DIFFERENTIAL DIAGNOSIS OF ACUTE APPENDICITIS, CHOLECYSTITIS, AND ASCENDING URINARY INFECTION

HISTORY

THE patient is an unmarried woman, aged forty-five. She entered the hospital March 30, 1914. Her family history is negative for tuberculosis. Menstrual history: Began at twelve years; always regular; every twenty-eight days until the age of eighteen; after that time every twenty-four days; ten days' duration; two napkins a day; no severe pain before the age of eighteen; since then always associated with severe, cramp-like pains and dragging pains in the right iliac fossa and in the lumbar region of the back. Three or four years ago she began to take from three to four drops of ergot daily, and since then the pain with her menstruation has been less marked. Since September, 1913, she has menstruated every twenty-eight days until her last period, which was twenty-three days ago. Then she began to menstruate today, March 30, 1914. She has no pain with this flow and has passed no clots.

When she was about eight years old she thinks she injured herself reaching while picking grapes. She says that after going home in the evening of that day she had dull, aching, dragging pains in the right iliac fossa and renal region. She went to bed, and hot applications were made over the area most markedly involved. She remained in bed about a week with a dull pain most of the time in the right iliac fossa. A doctor was called, but she does not remember what he said. He gave her some medicine internally. She continued tender for about three weeks. She knows nothing as to temperature or chills. Following this illness she had severe pains in the right iliac and lumbar regions when the least bit jarred. She often held her hand over her side to support it. She continued to have these attacks each fall at about the same time for about four years. Each time the attack lasted approximately one week, and she always had to go to bed and have hot applications made to her side. These

attacks were always followed by two or three weeks of local tenderness. Then she also protected her side with her hand while walking about. These attacks almost always followed wetting her feet. From the age of twelve to eighteen she was free from the attacks.

At the age of eighteen she began to menstruate every twenty-four days instead of twenty-eight, as heretofore, and developed a dull, aching, dragging pain again in the right iliac fossa and lumbar region, which continued all during her periods. A similar pain also appeared sometimes after wetting her feet or working hard. At other times she would be practically free from the pain, but was usually tender in this area.

She continued this way until three or four years ago, when she began to take three or four drops of ergot daily. The pains in the right side then gradually decreased in severity, so that she discontinued the use of the ergot two years ago. The pains were less marked during menstruation, and she could work with much less distress. About March 6, 1914, on the day of the onset of her menstrual flow, she wet her feet. The next day she began to have increased frequency of urination,—every fifteen or twenty minutes,—so that she had to wear a pad. She passed only a few drams at a time, but no blood. She used the pads until March 18, 1914, although the frequency of micturition had decreased slightly by that time. At the time she began to have the increased frequency of urination she developed a sharp, severe, burning pain, referred to the urethra and bladder, during urination. She had a dull pain in the region of both kidneys and up under both scapulæ. The burning pain in the bladder and urethra subsided March 15th, and has not reappeared. Since then she has had a dull, aching pain just along the costal margin down to the iliac fossa of the right side. There is also tenderness there. The condition is worse on some days than on others. Since March 17th she urinates three or four times a day and two or three times at night. She has never had a chill. Has had no pain on urination since March 15th. On March 25th her temperature was 101° F. Since then she has had about one degree evening temperature daily.

For the past fifteen years she has had belching of gas and eructations of acid fluid about one hour after meals. For the same length of time she has had severe, dull, aching pains in the epigastrium from about thirty minutes to one hour after meals. These pains last about one hour and are usually felt both anteriorly and posteriorly—anteriorly, about three inches below the ensiform cartilage and to the right of the median line. There is also an area of tenderness at the same site. The pains are always worse after large heavy meals and after eating meat and cold foods. They are less marked after eating warm foods. She never saw blood in the emesis. The pains are especially marked in the fall and spring of the year. She often has emesis of food from one to two hours after meals. This gives her immediate relief. She has also noted that if she eats a few crackers after the onset of the pain she gets relief for a time or until the next regular meal. All the symptoms have increased gradually up to the present time. She has never been jaundiced. Never has had chills, but frequently has had chilly sensations. Bowels are not constipated. Her appetite is always poor. She has lost little weight.

COMMENTS

DR. MURPHY (March 30, 1914): This patient told me she has been sick since the seventeenth of March, 1914. A doctor saw her late Thursday night, March 26th. She had been sick then for nine days. She is a woman of about forty-five, a nullipara, not married, and has no history of a vaginal infection at any time. She gives a history of repeated attacks of discomfort, some of them severe enough to be called pain, for a number of years in the right side. She was never jaundiced and never has had pain severe enough to require a hypodermic. On March 5, 1914, she was exposed to the weather and wet her feet. From that time she began to have increased frequency of urination both day and night, with great pain on urination and pain before and after urination. She continued having this difficulty for a number of days; then it diminished but did

not disappear. That is the story as she gave it to me. I will ask Dr. Tuite, who brought her here, to tell us his story.

DR. TUITE: I saw her late, perhaps 10 P. M., on March 26th. She was rigid all over the right abdomen, especially high up, and very tender. She had a temperature of about 101° F., if I remember correctly. The nurse said that the temperature had been up to 102° F. during the day. In the morning it was normal. I saw her again on Friday morning. She was a little less rigid over the abdomen, but still would not allow me to palpate deeply down there. Her temperature was 99° plus, but went up to over 101° F. that afternoon, the nurse reported to me. I saw her on Saturday morning again, and she was a little less rigid, but was having a great deal of pain. Sunday morning she was very rigid, the rigidity extending almost to the left side, but more in the upper quadrant. She was tender in the lower quadrant, but not at all so much as in the upper. Her pulse went up to 110 on Sunday. She vomited a day or two before I saw her, but she did not vomit with this immediate spell. In one spell she said she had attacks of "water-brash," but did not vomit. She said she had had similar attacks for years, but I do not know anything about the temperature in these attacks. She said she had urinary troubles in all the attacks. There was considerable pus in the specimen of urine I examined.

DR. MURPHY: Did she go to bed with the attacks?

DR. TUITE: She was in bed part of the time. She was up part of the day I saw her and she was running a temperature. The nurse had been with her only three or four days, and she had a temperature all that time.

DR. MURPHY: The doctor was called to see her on account of the increased severity of the pain on the right side and the increased illness, as interpreted by the nurse. The nurse insisted that she should have a doctor, and she reluctantly consented.

She has been catheterized this morning, and the report is twelve pus-corpuscles to a one-sixth field. That is too many for a *first* catheterized specimen. The question which comes up is,

where is that pus coming from? Is it from the cystitis which she gave evidence of in the early stage of this trouble—on the seventh of March? Is it from the irritation? Is it an extension of that infection up through the ureter, which is now sore and sensitive? The most sensitiveness in the abdomen at the present time is on the right side, in the loin. Is it an infection of the kidney? You can see that the only vomiting she had was on the seventeenth. She had no colic which was severe enough to require a hypodermic. She had been having this attack for at least three or four days before the doctor saw her. Let us figure out the condition which she had originally. Suppose it were an appendicitis beginning on the seventeenth of March—then she could have had an elevation of temperature before the doctor saw her. She could have had that increased sensitiveness as an extension of the infection from the appendix to the neighboring structures on the seventeenth. She could have a leukocytosis of 16,200 at the present time, all from an appendix; but she could not have these symptoms from an appendicitis that began on Thursday, because she had the temperature from Wednesday and it preceded the more severe pain. Is this more severe pain she has now the sequence of an extension of the appendicitis or is it an infection of the bladder extending up through the ureter into the kidney? Those are the two propositions. There is a third possibility. The pain and sensitiveness were not most pronounced at the point where the appendix is commonly found. They were at the point where the gall-bladder is located. They were most marked just under the costal arch. She had very severe pain under the right shoulder during these attacks. Did she have a gall-bladder infection? Was that trouble which she began to have on the 17th of March a gall-bladder infection, independent entirely of the cystitis? But she had a more pronounced elevation of temperature than is common with gall-bladder infections. A gall-bladder infection without a pronounced obstruction, as evidenced by colic or by dilatation of the gall-bladder, does not often give a temperature of 102° F. A gall-bladder infection with a small stone in the cystic duct, not moving, does not give a colic

at all. The infection which very often causes a high temperature goes with recurrent chills and fever, but, you see, that was not the type present here. The temperature wave here was not of the cholangitic type, but the wave of an ordinary retention-infection type. It went up to 102° F., was 101° F. last night, with a leukocyte response of 16,200 this morning. On examination today she is sensitive to perpendicular percussion under the costal arch. Then the question comes up, was this a kidney infection? Is it the kidney which you can feel distinctly when you make the examination? Is that sensitiveness, which you get from percussion, under the costal arch a kidney sensitiveness just beneath the abdominal wall? Then you should make the test for retention and infection in the kidney. Wherever you have retention and infection in the kidney, an obstruction to the ureter, or an obstruction to the pelvis of the kidney, make fist percussion on the back. We made that this morning and I am sorry I cannot make it before you. I first put my hand over the left kidney and she jumped. I said, "Did that pain?" She said, "No, you just frightened me." But she gave the typical result of no response when the test was actually made. That is why I have taught that you should make the test by first percussing over the normal side and then percussing with the fist over the supposed infected side. We then percussed over the supposed infected side and she gave no response at all, no evidence of pain at all, which makes one question the kidney as the source of this trouble, notwithstanding the twelve pus-corporuscles in a field. Now the question comes up, is it the appendix or is it the gall-bladder? The clinical history is against an appendicitis. The disease did not begin like an appendicitis. It did not act like an appendicitis. It has not the characteristic sensitiveness of an appendicitis now. The history and the sensitiveness to percussion on deep inspiration under the costal arch look more like an infection of the gall-bladder; but the acute symptoms are subsiding, we believe. We shall make a leukocyte count, and if it is still going down, we shall wait. If not, we shall operate tomorrow. We shall examine the urine and see if it still shows pus. She had pain under the

scapula, and pain under the scapula is indicative of gall-bladder disease, and was so interpreted many years ago by Frerich. He was fairly right in his interpretation of that sign when associated with other things. At one time, when we were draining the gall-bladder frequently, as that was the treatment for cholecystitis as well as cholelithiasis then, we tested out a number of these cases in series of tens. We came to this conclusion: when we had the ureteral sound in the neck of the gall-bladder, seven times out of ten the pain was referred to the right scapula. The next place in point of frequency was the epigastrium. The next in frequency was under the right or left clavicle. Seven times out of ten it was felt under the scapula on the right side, which shows that Frerich had interpreted that sign about right.

I think it looks as though we were justified in waiting here without arriving at a positive diagnosis between these three lesions. There is no evidence of a pelvic infection at all. The pelvic pouch moves around perfectly freely and she has no pelvic pain. We may exclude an ascending infection from the vagina. It is just the right period of time—from ten days to two weeks—for the primary infection of the bladder to develop into an ascending infection of the ureter, with some discomfort and frequent urination. We shall cystoscope her tomorrow and if it is the kidney which is involved, the corresponding ureter opening will be red, congested, and edematous. The left ureter will not be red and congested; we must come to our conclusion by excluding things in a systematic way, so as not to go in and take out an appendix and not to open the gall-bladder when the lesion is in the kidney. She may have a chronic appendicitis running back all these years and giving her recurrent attacks, as interpreted by the doctor, with a secondary infection of the gall-bladder, which is one of the common combinations which we meet with.

[Bacterial examination of this patient's urine revealed a pure culture of *Bacillus coli*. This patient was not operated, but an auto-vaccine was made from the colon bacilli and administered. She improved rapidly. Her pain and pus disappeared. She left the hospital April 18, 1914.—ED.]

CARCINOMA OF THE STOMACH AT THE CARDIAC ORIFICE. DIAGNOSTIC TALK BY DR. CHAS. L. MIX

HISTORY

PATIENT, male, aged fifty-four, entered the hospital February 26, 1914, with the following history:

Family history: No tuberculosis or carcinoma. Past illnesses: Was jaundiced twenty-eight years ago; had no pain; jaundice lasted for four days. Had measles.

Present illness: About February 1, 1913, he first noticed difficulty in swallowing coarse or poorly masticated food. It seemed to him as though the food became impacted in the esophagus low down, about the level of the ensiform cartilage. Sometimes after eating coarse food he would regurgitate it almost immediately as if its passage into the stomach were blocked. He immediately would feel relieved. He had no distinct pain at that time.

Since about January, 1912, he frequently has had emesis of a mucus-like, rusty material. He would vomit such material three or four mornings a week. He then would have no more emesis during the day. He felt severely nauseated on getting up in the morning, always before breakfast. The patient went along in this condition until about March, 1913, when he first noticed blood in the vomitus. At that time he vomited about two ounces of pure blood. Immediately following this emesis he began to have pain in the epigastric area—sometimes a burning, cutting pain, at other times griping. This pain would begin immediately after he started to eat and would continue for an hour after eating. It was not relieved by drinking hot or cold water. He belched no gas at this time. His appetite was very poor. He had no preference for or dislike for any kinds of food. He has had in all about twelve emeses of blood up to the present time. He has never seen pure blood in the stool. He has had dark stools occasionally, but no tarry stools.

He gradually grew somewhat worse from March, 1913, to November, 1913, when he went to a hospital. He was put to

bed and given special diet. The doctor made a physical examination, took a skiagram, and said that he might have an ulcer of the stomach. No test-meal was taken. He remained in the hospital two weeks, then was given a prescription and permitted to go home. Since that time he has grown progressively worse up to ten days ago. He has had pain almost continuously of late. He also has had regurgitation after eating anything coarse. He could eat soup without regurgitation, but sometimes has distress and pain. He has lost about 30 pounds in weight. Ten days ago the doctor passed a stomach-tube and drew off a test-meal with large quantities of blood in it. He has felt much better since then. He has had no emesis since and has been able to eat more coarse food than previously.

COMMENTS

DR. MIX (February 28, 1914): The history, perhaps, can be stated a little more clearly. Here is a man who is a saloon-keeper and has been in the habit of living as most saloon-keepers live for quite a number of years. Beginning with January, 1912, something over two years ago, he had the typical morning vomiting of the alcoholic. We find stated in his history that he vomited up a mucus-like, rusty material three or four mornings a week. We find that the vomiting took place in the early morning, before breakfast; that it was associated with nausea on rising. This symptomatology points very clearly to a chronic alcoholic gastritis; it may well have been the chronic irritation that has led to the malady from which he is now suffering.

After having this occasional morning vomiting for a number of months he states that, beginning with February, 1913, about a year ago, he had difficulty in swallowing coarse food and food which he had not sufficiently masticated. He states very definitely and clearly that the food seemed to go down all right, but that it got stuck somewhere and made him very uncomfortable until he got it up again. He had no definite pain with this sensation, but merely the sensation of obstruction; and in illustrating its location, he placed his hand below the left costal border across the stomach. He then went further and said

that it seemed to him as though the food went into his stomach, but that it could not go through the stomach and get out.

About a month after he had noticed this difficulty in swallowing he vomited up about two ounces of blood. Since March, 1913, he tells me that he perhaps has vomited blood in similar amounts at least a dozen times.

Beginning with March, 1913, the pain started. This pain was located in the epigastrium, and it was associated with a feeling of burning and gnawing, sometimes griping, as though there was some constriction somewhere. To me a griping pain usually means a contraction of the circular muscle-fibers somewhere in the alimentary canal, and such contraction ordinarily is the result of some local pathology of serious danger. We find further that this pain came on immediately after eating, and, indeed, while he was eating, and that it lasted for an hour or more. So clear is the statement in this regard that one can feel certain that eating caused the pain. Furthermore, drinking either hot or cold water did not relieve the pain, and the pain was actually aggravated by the taking of additional food. His appetite became poor, and there was no preference or dislike for special articles of food. Very noteworthy is the fact that he did not complain of gas or of belching. It was noted further that there was no history of tarry stools.

In November, 1913, things had come to such a pass with him that he thought it advisable to go to a hospital and have a diagnosis. Here it was believed that he probably had an ulcer of the stomach, the diagnosis being based upon the history of pain immediately after eating and the vomiting of blood, without a careful consideration of the history as a whole. In this conclusion you will see many errors. The pain, instead of coming on immediately after meals and lasting for an hour or two, lasted all the time, and he regurgitated everything he ate except soup. His last venture before coming to the hospital was to see a physician, who concluded, on account of the alcoholic history, the morning vomiting, associated with the vomiting of small amounts of blood, that the patient must be suffering from cirrhosis of the liver. The patient has lost 30 pounds in

weight. The loss of weight was looked upon as certainly confirmatory of that diagnosis.

Let us now analyze these symptoms. The salient feature in his case is the vomiting of small amounts of blood, never more than two ounces at a time, on at least twelve different occasions in the last year. The diagnosis is betrayed in this point. In ulcer of the stomach there is one or possibly there are two big hemorrhages. Usually there is but one hemorrhage, large in amount. Little hemorrhages of small amounts do not take place in ulcer of the stomach.

The same is true of cirrhosis of the liver. In those cases of atrophic cirrhosis of the liver with portal blockade, there is an establishment of a collateral circulation between the gastric or coronary vein and the esophageal veins at the cardiac orifice, and there are usually developed a series of esophageal varices. These varices not infrequently rupture and fill the stomach with blood, which is vomited up. Small repeated hemorrhages over many months do not occur in cirrhosis of the liver.

There is only one pathologic process which is associated with small repeated hemorrhages. Take, for example, the history of carcinoma of the uterus—what do you find? Not one big hemorrhage, then none, but rather a repetition of small hemorrhages over a long period of time. What do we find in cases of carcinoma of the stomach? Not one large hemorrhage, but rather more or less continual pouring out of small amounts of blood, separated by intervals of only a few days or weeks, with coffee-ground vomitus. Wherever carcinoma occurs, whether in the uterus or rectum, stomach or esophagus, the same law of hemorrhage will be found to obtain. Cirrhosis of the liver or ulcer of the stomach with a history such as is found in this man's case cannot be diagnosed. The history is absolutely against it. True, this man is a saloon-keeper. He has been in the habit of drinking altogether too much alcohol, and further he had chronic vomiting, but his vomiting and his bleeding are not due to this cause.

Examination of the abdomen in this patient's case shows no abnormality except one thing. In the epigastrium, on deep

palpation, one meets with a feeling of rather marked resistance. You will note that I cannot get my finger down in that area [indicating the area to the left of the epigastrium beneath the costal border]. Furthermore, there is a feeling of tenderness when I press down upon that area. This tenderness might suggest some perigastric irritation. I wish to call your attention to the well-developed man he is. He looks well nourished, notwithstanding his history of losing 30 pounds in weight. You see his color is good. I wish to emphasize the fact that his general appearance is, on the whole, excellent, and that by merely looking at him one would scarcely suspect the presence of carcinoma.

Now he was told that he had cirrhosis of the liver. Please observe that the liver in his case is not atrophic or enlarged. Please note also the absence of a collateral circulation in his flanks. I rarely make a diagnosis of atrophic cirrhosis of the liver without the evidences of the establishment of a collateral circulation. In his case there are no signs of the establishment of such a circulation about his umbilicus, about the rectum, or in the sides, these being the points where such a collateral circulation is usually established.

The patient was also told that he had an ulcer of the stomach, and with a good deal of reason. He has the history of the vomiting of blood, and, in addition, there is the history of an irritation of the stomach by food and the regurgitation of food.

Let us now analyze the statements. We find that beginning with January, 1912, he had a chronic gastric irritation, due undoubtedly to the alcoholism, associated with chronic nausea and vomiting before breakfast. To my mind the alcohol led to the trauma which was responsible for the later development of his symptoms. In February, 1913, his first difficulty in swallowing presented itself. He found that he could not swallow coarse food or food that was poorly masticated. It seemed to go down, as he expressed it, and to stick in his stomach, where it disturbed him until it was regurgitated. There was no distinct pain in the first month of his trouble, but in March, 1913, after the difficulty in swallowing had persisted a month, he

vomited a couple of ounces of blood, and with the denudation of the raw surface, evidenced by this hemorrhage, pain appeared, located in the epigastrium, of a burning, gnawing, griping nature. The burning and gnawing character of the pain indicates a denuded surface irritated by the passing of food. Griping to my mind indicates a contraction of the circular muscle-fibers. The history thus far is very suggestive of a neoplasm blocking off the entrance of food into his stomach, with the ulceration of the neoplasm by March, 1913. The neoplasm undoubtedly began before February, 1913, but during that month attained a size sufficiently great apparently to blockade the esophagus. In March, 1913, the ulceration of the neoplasm led to the pain which came on immediately after meals and lasted for an hour or two, until the effects of the irritation of the passing food had subsided. Of course, hot or cold water had no effect in relieving the pain, and, of course, gas would not be present in a lesion of this character. There are, however, a loss of appetite and weight, such as he shows, and the absence of jaundice and tarry stools.

He got along through the summer indifferently well, occasionally vomiting blood, and finding more difficulty in eating, until he finally went to the hospital in November, 1913, where the diagnosis of ulcer of the stomach was made, although no test-meal was given, and although the history clearly indicated the extreme probability of a carcinoma at the cardiac orifice. Since November his condition has grown steadily worse, until now he regurgitates pretty nearly everything except soup.

Very significant is the fact, stated in the history toward the end, that ten days before entering the hospital a physician passed a stomach-tube and drew off a test-meal with large quantities of blood in it. As a matter of fact, the patient tells me that the doctor did not get any of the test-meal—that practically all he did get was some blood in his stomach-tube. Curiously enough, the patient has felt much better since that time. He has had no vomiting since then and has been able to eat more coarse foods. Why? Because the physician pushed his stom-

ach-tube down through the carcinoma and produced a temporary opening into the stomach.

How do we know this man has a carcinoma? For a very simple reason. A test-meal was given to him previous to his coming to the hospital and we did not give one. The reason we did not give one was because the physician who previously attempted to pass the stomach-tube in our opinion failed and merely shoved it into a carcinomatous mass. We, therefore, decided to locate, if possible, the area of obstruction, and for that purpose very carefully passed a stomach-tube. The stomach-tube went as far as the cardiac orifice, where it stuck. On withdrawing the stomach-tube we obtained from the eye of the tube a very small piece of tissue. This tissue, which I was careful to look for and which was freshened out in some water immediately on withdrawing the tube, was examined microscopically and found to be cylindric-cell carcinoma. Such a cylindric-cell carcinoma is usually located at the junction of the esophagus with the cardiac end of the stomach. Had this tube been passed by a nurse, the piece of tissue would have been overlooked. These little pieces of tissue are exceedingly valuable, making unnecessary at times exploratory laparotomies. Shreds of tissue which are obtained, though extremely small, are often sufficient. Dr. Zeit, who examined the tissue, told me last night that he could be "cock-sure" about it. I think any one who looks at the slide will at once declare that it is a beautiful specimen of a cylindric-cell carcinoma from the cardiac orifice of the stomach.

The patient is up and walking about, and in apparently good health. He will not, however, long remain in this state. The loss of weight in these cases is extremely rapid, not only on account of the carcinoma itself, but on account of the associated starvation.

There is one lesson in this case which is of exceptional value, that is, that the vomiting of small quantities of blood for a long period of time does not belong in the history of either gastric ulcer or cirrhosis of the liver; and it is a very strong indication of the probable existence of a carcinoma at or near the cardiac

orifice of the stomach, for if the carcinoma were in the stomach, the vomiting would be coffee-grounds in color.

[The opening in the stomach will be kept open as long as possible by the careful use of graduated bougies. If the opening closes or the bougies cause bleeding, a gastrostomy will be performed.—Ed.]

TENOPLASTY; TENDON TRANSPLANTATION; TENDON SUBSTITUTION; NEUROPLASTY

COMMENTS AND CASES

DR. MURPHY: Tendon transplantation as substitution for lost motion in paralyzed muscles is a very promising field of work. We have had a rather unusual opportunity to study such cases. Many of the late results of anterior poliomyelitis, and also of injuries—ischemic myositis, for instance, of which we have often spoken here—come to this clinic. The results which we have obtained in many of these cases have exceeded our fondest hopes of a few years ago. Tendon substitution by the use of silk or other foreign material has also met with success elsewhere; but we have had no experience with it for the reason that we prefer autoplasmic means which effect the same end with greater ease and certainty.

What I wish to dwell on particularly today is the technic of tendon transplantation for the purpose of supplying lost motion in opposing muscle-groups, irrespective of the origin of the paralysis and the surgical principles underlying the procedure. Some of these cases have been due to anterior poliomyelitis, some to encephalitis, and others to disease of or division of the peripheral nerve-trunks.

In the treatment of all these paralyses a careful search must be made for the muscles retaining their contractility before a final decision can be made as to which tendon or tendons shall be transplanted. When this information is definitely obtained, one next must take into consideration the desirability and advisability of producing an arthrodesis in one or more of the joints in the impaired extremity instead of a tendon transplantation. Arthrodesis combined with transplantation of muscle attachments often produces an effective restoration of

function of the limb, and restores a limb, which has been comparatively valueless, to useful service. Arthrodesis sometimes may be preferable to tendon transplantation, but both of these procedures are secondary in importance to restoration of the continuity of the nerve axon, when possible, so as to restore muscular contractility to the paralyzed muscles. In some cases transplanting a degenerated distal nerve-trunk into the proximal end of a living nerve may effectually restore motion. (See December, 1912, CLINICS.)

In the paralysis following anterior poliomyelitis, which is the most common lesion causing regional muscle paralysis, the secondary motor neurons are often so extensively damaged that the switching over of adjacent motor impulses cannot be effected. The field in which this "switching" or "bridging" operation is most valuable is in paralysis of the external popliteal nerve without paralysis of the internal popliteal. In such a case the axons of the external popliteal nerve can be inserted into the axons of the internal popliteal with great advantage to the patient.

Next in importance are the paralyzes of the muscle-groups of the arm, particularly those innervated by the ulnar and median nerves. When one or the other of these groups is paralyzed, an implantation from the other nerve may effectually restore function. Paralysis of the median nerve is the more important affection. The nerve implantation should be made months before the tendon transplantation is attempted, so that, if possible, there may be nerve restoration and corresponding muscular contraction present before the tendon transplantation is carried out. Unfortunately, too many of the cases of paralysis due to anterior poliomyelitis are discarded or overlooked as surgical possibilities, and braces and other substitutes for tendon transplantation are adopted instead. There is a broad field for the application of these principles of operative surgery in this class of cases, a fact of which the general practitioner should be made aware.

It is frequently necessary, for instance, where a contracture has been permitted to increase gradually for years and the leg

has become badly deformed, to divide and elongate the two hamstring tendons and possibly cut also the posterior capsule of the knee. If the contracture has existed for only a comparatively short time, capsulotomy is unnecessary, although tenotomy and tenoplasty may be demanded. In all such cases the capsule, the tendons, and the remaining tissues should be so fully elongated under anesthesia that the limb lies in a perfectly straight position and can be put through its full normal range of movement without great force or tension; otherwise, when the patient comes out of the anesthetic, motion still will be limited and deformity will persist.

It is remarkable what extremes of contracture may take place as the result of complete division of the bellies of large muscles. If the gap is too great to bridge over or the muscle-ends thoroughly fixed to the periosteum, full restoration of function cannot take place. When there is, however, merely a deep depression in the belly of the muscle following suture, or after subcutaneous union without suture, the full strength of the muscle is often restored. Frequently the upper or lower portion of a muscle, following a compression myositis, hypertrophies greatly and renders wonderful service, considering that only a small percentage of its contractile cells remains intact.

I had a patient, a professional athlete, who suffered a traumatic division of his right biceps. He had one of the largest biceps I ever saw. The injury was caused by a railroad accident. He agreed that if his muscle power was restored to its full lifting capacity, he would exact no damages from the railroad company. Eight months after the accident, when he would contract his biceps, my fist could be inserted into the gap between the two ends of the muscle; and yet he could lift with that biceps a greater number of pounds than he was able to lift before the accident. It was a subcutaneous transverse division, and had not been united by suture. There was subcutaneous spontaneous reunion.

Wherever the tendons have been destroyed and there is not sufficient tendon material left for proper elongation, autotransplantation should be done. Portions of tendons may be

taken from almost any of the other tendons in the patient's body, the Achilles tendon being an excellent source of supply. The long flexor tendons of the forearm may have some of their substance removed and yet not have their function impaired in any way. The relatively unimportant palmaris longus tendon may be used in its entirety. These same flexor tendons may be used as substitutes for ligaments in flail joints or in almost any joints with excessive motion, but particularly in the knee, where substitutes for the external lateral ligaments may be necessary following arthroplasty.

Silk sutures may be used as ligament producers or stimulators. They can be inserted so as to induce around them the development of a mass of connective tissue which will give added mechanical support to a joint; or, if used as a tendon, they will favor the generation of a sufficient quantity of connective-tissue fibers to carry on the function of a tendon. When the silk ceases to give support, it is no longer of use *per se*.

The after-treatment of a tenoplasty consists, first, in waiting until the wound has healed entirely, and, second, in gradually instituting passive and active motion. After tendon elongation active motion first should be resorted to, as the patient thus enables us best to estimate pain and discomfort, and the amount of muscular contraction which the impaired tendon will tolerate. At the end of from four and a half to six weeks, when an organic union of the tendon has taken place, passive motion may be instituted vigorously and must be encouraged. During the process of repair of the tendons, the limb should be fixed in a plaster mold, wire-gauze frame, or by some other mechanical support, in an overcorrected position, so that any fixation of the tendons which may occur transversely to their axes will be fixation with ample elongation. When muscles have contracted and atrophied for a number of years, the restoration of voluntary contractile power in them takes months.

The fascia lata has also been transplanted for the purpose of supplying new tendons, in fact, fat and fascia from any portion of the body may be used to lengthen a tendon and in that way overcome a very evil result, namely, the contraction of a muscle

to a degree which interferes with its voluntary lengthening and, therefore, produces a lesion which, while apparently, for instance, a paralysis of the extensor group of muscles, is in reality merely a contraction of the flexor group, as in the ischemic myositides.

There are many methods for lengthening tendons; in fact, almost every experienced operator has devised or modified some method which gives him good results, and which he, therefore, prefers to any other. The principle involved in all the methods is a splitting of the tendon in such a way as to lengthen it. We use what we call the "double L" method of cutting and splitting. It is easily done and does not impair the vitality or integrity of the tendon.

The particular method of tendon transplantation for the

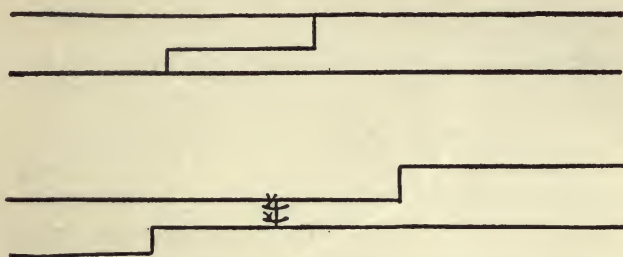


Fig. 153.—The "double L" method of cutting and splitting the tendon for tendon elongation.

restoration of lost function in a muscle or group of muscles which we employ preferably is the so-called Vulpus method. The entire half of the tendon, or the portion which has been partially split off, is sutured to the tendon or muscle which it is intended to strengthen. In order to show you precisely just what work we have done here, I shall take up a few illustrative cases.

The first patient I shall refer to was a doctor on whom we operated twice, once for the purpose of giving him a serviceable pseudoarthrosis in his lower arm. The humerus was fractured, with non-union just above the elbow, and the elbow was ankylosed. This was a shotgun wound, fracturing the lower end of the humerus, infecting the elbow-joint, and carrying away several inches of musculospiral nerve. We could not perform

an arthroplasty with pedicled fascia and fat flap in that case, as the neighborhood of the whole joint was scar tissue. Therefore we made him a controllable movable joint at the site of the non-union of the humerus fracture. The result was a good one. (See CLINICS, vol. ii, p. 651.) He also had a paralysis from division of his musculospiral nerve and a consequent wrist- and finger-drop. At the time of the operation on the arm we could not find the ends of the nerve because of a mass of scar tissue enveloping them, and, therefore, we decided to perform later a tendon transplantation to restore extension function in his hand, thumb, and fingers. The original operation on his arm was performed in April, 1913; and on December 1, 1913, we operated for the paralysis of the extensor muscles of his hand and fingers. What he wanted particularly was the ability to use his hand in obstetric work, that being his specialty. His flexor tendons were all in good condition because his median nerve was intact, but his extensor muscles were not functioning because of the division of the musculospiral. The propositions to be met were these: He must be able to throw back his hand and extend his fingers. We dared not take away too much of his flexor muscle power, and yet we had to supply efficient extension.

The plan adopted in that patient was one which we had used in previous cases with good success, namely, to convert the flexor carpi radialis into an extensor muscle by transplanting its tendon into all the extensor tendons of the thumb and fingers. He could easily spare his flexor carpi radialis because the loss of its flexor function would not interfere with finger flexion and would still leave him wrist flexion, so long as the ulnar carpal flexor and the palmaris longus were functioning. In a very short period of time he could train his flexor carpi radialis to throw back and straighten his fingers. It was absolutely impossible in that patient to do anything with the musculospiral nerve, and, therefore, there was nothing left for us to do except this tendon transplantation. He could pronate and supinate, and he was able to flex his arm as the result of his pseudoarthrosis so that he can now touch his nose and the top of his head with his fingers.



Fig. 154.—Old compound fracture of humerus just above elbow, with non-union. Bony ankylosis of elbow, musculospiral paralysis with wrist-drop. Too much scar tissue about the elbow to permit a fascia and fat flap arthroplasty. The *x*-ray shows the deformity before the operation.



Fig. 155



Fig. 156

Figs. 155 and 156.—Old compound fracture of humerus just above elbow with non-union. Bony ankylosis of elbow, musculospiral paralysis with wrist-drop. Too much scar tissue about the elbow to permit a fascia and at flap arthroplasty. Front and lateral views of the deformity.



Fig. 157.—Old compound fracture of humerus just above elbow, with non-union. Bony ankylosis of elbow; musculospiral paralysis with wrist-drop. Rear view of the deformity before operation.



Fig. 158.—Formation of a new joint (pseudoarthrosis) at site of old fracture. Considerably more flexion than indicated by the roentgenograms was possible after the formation of the pseudoarthrosis.

We made a longitudinal incision on the anterior surface of the wrist, close to the radial side, and just internal to the radial artery, so that the flexor carpi radialis tendon was easily brought into view. We were very careful in making this dissection not to injure the blood-vessels or nerves. The latter are readily distinguished by their buff color; the tendons are white and shining or glistening. The flexor carpi radialis tendon was divided close to the tubercle of the scaphoid, and the end of the tendon was grasped with a hemostat.

A second incision was now made over the dorsal surface of the wrist, exposing the extensor tendons. The proximal end of the flexor carpi radialis tendon was dissected back three inches and was carried through a tunnel underneath the skin and brought out through the second incision, in its passage transfixing the extensor pollicis and extensor indicis tendons. The flexor carpi radialis was then split lengthwise into two equal portions, one half being carried anterior to and the other half posterior to the extensor tendons of the other fingers. The divided tendon was then tacked not only to the extensor pollicis and extensor indicis, but also to the common extensors with chromicized catgut sutures so as to secure good fixation. The wounds were then closed, and the hand was dressed in extreme extension and held in that position by means of a wire gauze splint placed on the anterior surface of the arm and hand.

The case progressed very satisfactorily. The wounds healed by primary union, and within two weeks after the operation the patient began to have some extensor motion of his fingers. He left the hospital about four weeks after the operation with very good extensor motion of his hand. We saw the patient quite recently,—about three months after the tendon transplantation was done,—and it was a pleasure to see how well he was able to use his hand. He could extend the hand and wrist in a straight line with the forearm, and his flexion was in nowise impaired. He was well satisfied with the result and so were we.

A second very similar case, operated more recently, was the following: Patient, a boy of nineteen years, entered the hospital January 21, 1914, with the following history: On February 14,

1913, at his home in one of the Southern States, he sustained multiple fractures of his right humerus. He was taken to a hospital, where reduction was attempted under anesthesia and a splint was applied, which was kept on for nine weeks. He returned to the same hospital in April, 1913, and skiagrams showed multiple ununited fractures of the humerus at its lower third. A Lane plate then was put on—in April, 1913. A splint was applied and kept on for eight weeks. The day after the operation he felt a numbness in his fingers. On removing the splint at the end of eight weeks it was found that he had a wrist-drop. October 18, 1913, we operated on his arm mainly for the purpose of determining the extent of the injury to his musculospiral nerve. We found that it had been severely traumatized at the site of the fracture, and that the axons had been destroyed by compression in a cicatrix for a distance of about three inches. The nerve was nodular and hard. A resection of the destroyed part and a neuroplasty were out of the question, because of the distance to be spanned. Something had to be done for him, however, because he had the drop-wrist. We explained the situation to him after he regained consciousness and suggested that he wait a few months and see whether the operation done to relieve the compression of the nerve by the cicatrix would benefit him in any way so far as regaining any power in his musculospiral distribution was concerned. We had freed the nerve from the external compression by the cicatricial tissue and kept in mind the possibility that there might be some return of function, although when we applied the faradic and galvanic current to the nerve above the site of the injury we failed to get a response in the muscles of the forearm or hand, proving conclusively that there were no axons to transmit impulses.

Complying with our suggestion the patient returned and was operated February 4, 1914, for the purpose of giving him some extension in his hand. I want to emphasize in connection with this patient the fact that his was not a doctor's lesion. It was a traumatic affair; the nerve-fibers were destroyed by a distinct and definite trauma other than that which might come from a tight bandage or a badly applied splint.

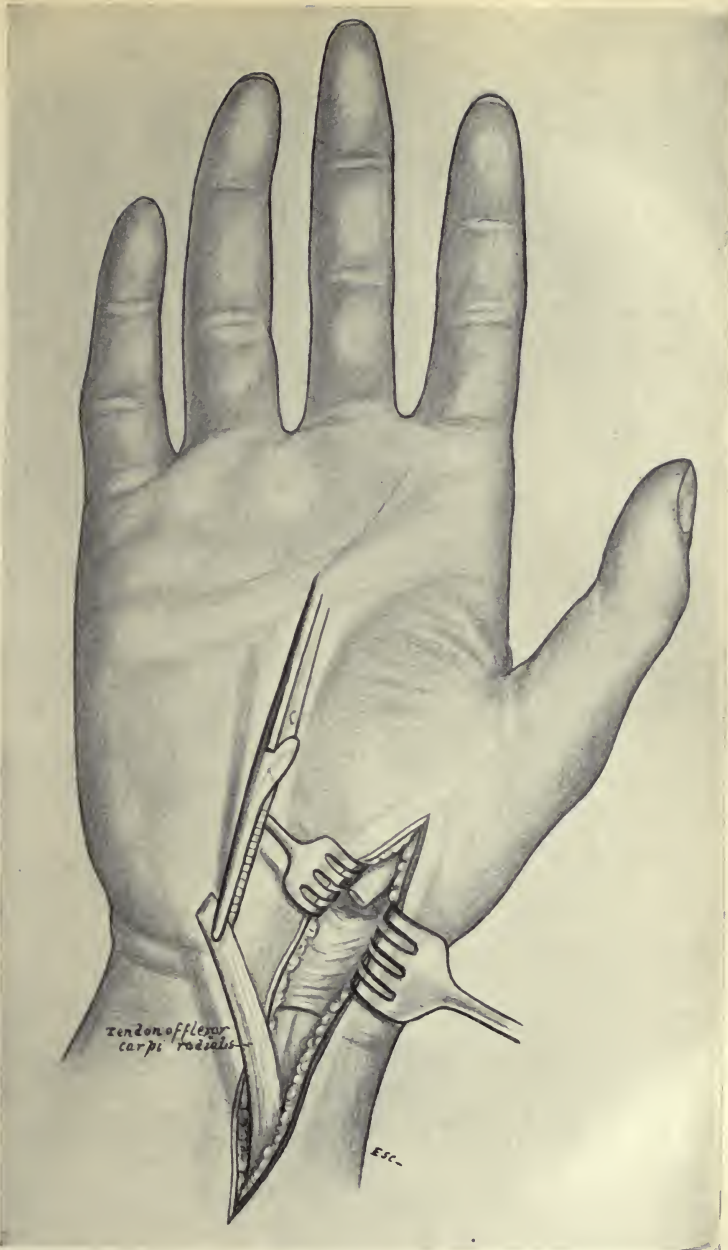


Fig. 159.—Method of transferring the flexor carpi radialis to the back of the hand to serve as an extensor of all the digits.

Step I: Freeing the flexor carpi radialis at its insertion, preparatory to passing it under the skin to the back of the hand.

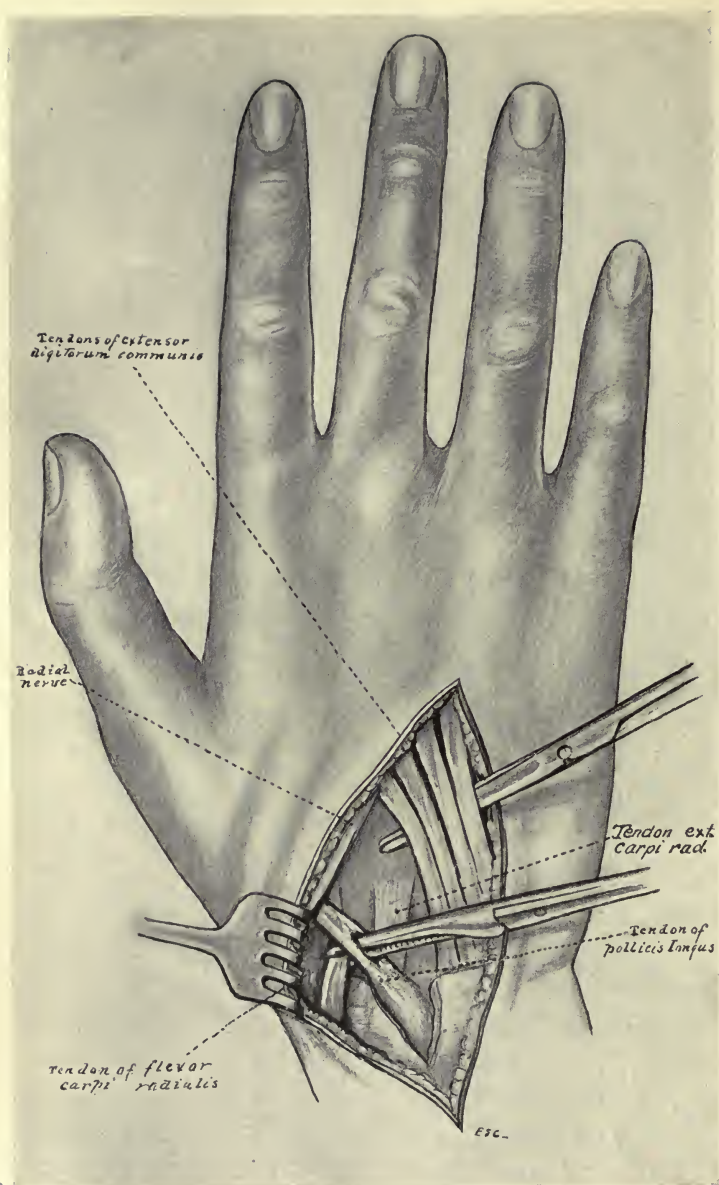


Fig. 160.—*Step II*: Exposure of the extensor tendons of the thumb and digits. Transfixion of the extensor pollicis longus tendon by the tendon of the flexor carpi radialis.

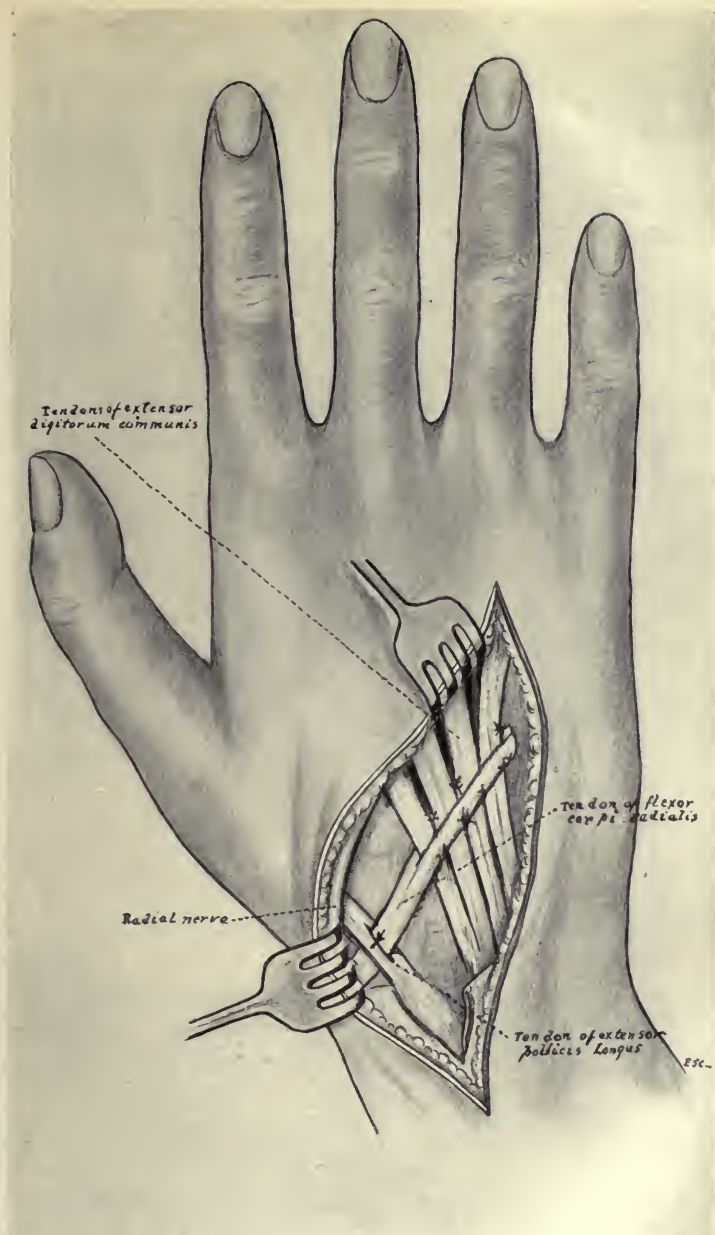


Fig. 161.—*Step III*: Method of tacking the split flexor carpi radialis tendon to the front and rear of the extensor tendons of the digits.

Now, then, what were we to do? There were two lines of available procedure. One was to do a neuroplasty on his musculospiral nerve, and the other was to supply him with some sort of extension of his thumb and at least two of his fingers, so that he could use his hand. As stated above, the neuroplasty was out of consideration because of the enormous distance which had to be spanned. Therefore, the best plan—and it was the only one seriously to be considered—was to transplant some of his flexor tendons and convert them into extensors. Of course, we did consider the possibility of transplanting the distal end of his musculospiral into his median nerve below the point where the latter gives off its main branches to the long flexors; but that was a physical impossibility, as you will note by looking over your anatomic charts. We did not dare transplant it into the median above the elbow, lest we destroy some of the axons to the important flexors. Therefore, we returned to our original decision that tendon transplantation would be the best procedure for this patient. If we could successfully have carried out a nerve transplantation, we should have preferred to do it, because nerves learn to change their function more readily than do muscles. We found that to be a fact when we made our original experiments on nerve-crossing in dogs. After the operation the dogs were a little awkward at first in moving their legs in the proper direction, but within a week or two they got along very well, showing that we had succeeded in getting the change in function which we desired.

The patient whose case we now are considering had considerable atrophy of the muscles of his forearm, but we were hopeful that we might be able to get a good result for him. The procedure we adopted in this case was similar to that employed in the previous case. We freed the flexor carpi radialis tendon close to its insertion into the scaphoid, and swung it back under the skin into the dorsum of the hand, passing through and transfixing the extensor pollicis and the extensor indicis tendons, and then sutured it to the posterior surface of the common extensor, spreading out the end of the tendon as much as possible in order to get a good attachment. The tendon was also tacked to the ex-

tensor indicis and the extensor pollicis tendons; so, if our plan was as successful as we hoped it would be, he would have good motion in at least the thumb and two of his fingers,—the index and middle fingers. The skin wounds were closed with horsehair and the hand was dressed in extreme extension, the position being maintained by means of an anterior wire-gauze splint.

The case progressed very satisfactorily, the wounds healing by primary union. When he was examined again two weeks after the operation, we were gratified to see that he had excellent extension of his fingers. The result was really remarkable, for an operation performed only two weeks previously. Even though so short a time had elapsed since the operation, it would have been almost impossible for one not familiar with the case to guess what was wrong with this patient before the last operation, as he showed no longer any evidence of the musculospiral "drops." He now flexes and extends his fingers with ease, moving them all, including the thumb. His grasp, of course, was in nowise impaired. We believe that this result was the best we have obtained in any of these cases. This case was exhibited to the visiting members of the International Surgical Congress on April 22, 1914, and they were unable to make a diagnosis of the previous musculospiral paralysis, his hand functioned so perfectly.

I might mention in this connection the case of a man on whom we operated about a year and a half ago. His history was as follows: Patient, male, aged twenty-two years, entered the hospital September 24, 1912, on account of paralysis of the left forearm and hand. His trouble began when he was two years of age. One day his mother noticed that her child did not use his left arm and that he cried when any one touched him. A doctor was called who said there was a fracture of the forearm near the elbow. There was no history of trauma, however. Since then he has been able to execute only certain limited movements.

Examination showed that the arm was not fully developed. The forearm was habitually held in supination, with the wrist fully extended and deflected toward the radial side. The muscles of the front of the forearm and of the thenar and hypothenar eminences were atrophic. Voluntary pronation and supination were



Fig. 162.—Tenoplasty on the wrist. Transplantation of the flexor carpi radialis into the extensor digitorum, extensor indicis, and extensor pollicis longus in order to restore to the hand extensor function lost as the result of a supracondyloid fracture of the humerus, with involvement of the musculospiral nerve in the callus. Power of complete extension regained as the result of the operation.



Fig. 163.—Tenoplasty on the wrist. Transplantation of the flexor carpi radialis into the extensor digitorum, extensor indicis, and extensor pollicis longus, in order to restore extensor function to the hand, lost as the result of a supracondyloid fracture of the humerus, with the involvement of the musculospiral nerve in the callus. Power of complete extension regained as the result of the operation. Power of flexion of the fingers and wrist not noticeably impaired by the dorsal transplantation of the flexor carpi radialis or by the other steps of the operation.



absent. He had full power of flexion of the elbow, but limitation of extension. He also had full power of extension of the wrist and of the small joints of the fingers, but no power of flexion. There was no paralysis of the upper-arm muscles, but they were somewhat paretic and small. The flexors on the front of the forearm, the adductors and abductors and *opponens pollicis* in the hand were paralyzed, also the extensors of the thumb. He had almost full power of extension of the three inner fingers. No loss of sensation.

October 8, 1912, a dorsal incision was made over the first metacarpal bone, and the tendons of the *extensor carpi radialis longior* and *brevior* were exposed and cut and a stitch was put in the end of each to keep them located. A second incision was then made over the outer side of the radius, where the radial nerve crosses around and the tendons and muscles were freed and drawn out. A third incision was made over the front of the wrist and the common flexor tendons were exposed. One *extensor carpi radialis* tendon was drawn under the short extensor tendon of the thumb and sutured to the *flexor longus pollicis*. The other was sutured to the flexors of the index and middle fingers.

The wound healed by primary union, and slowly but surely the patient began to regain the power of flexion in his fingers. Notwithstanding the long period which had elapsed since the onset of his trouble, about twenty years, we still were able to convert these extensor tendons into flexor tendons. We had the patient hold a roller bandage in his hand, so that he would have something on which to flex his fingers. A curious feature in the case was the fact that the patient was unable to flex the fingers of his left hand—the operated hand—without at the same time flexing the fingers of his right hand. He could flex the fingers of the right hand without flexing the fingers of the left hand, but when he tried to flex the fingers of the left hand he invariably would flex the fingers of his right hand. Such so-called “associated movements” are well known to neurologists as a congenital affection; but we know of no similar instance of the acquired variety.

The patient left the hospital about two months after the op-

eration, and he had at that time sufficient power of flexion in his hand to hold the roller bandage quite securely.

A somewhat similar case, involving the lower extremity, was the following: Patient, female, aged nineteen, had poliomyelitis fifteen years ago, when she was four years old. We could not get a definite account of the original trouble, so that we were obliged to base our diagnosis on a rather meager history and the present condition of the patient. It appeared that the girl at the time of her acute attack was perfectly well in the afternoon, but that at night she developed a fever and the following day became delirious. When she regained consciousness two days later she had an almost complete paralysis of the right leg.

When she entered the hospital the foot was inverted, and she was unable voluntarily to evert it. She had power in the muscles attached to the tendo Achillis, and to a slight degree in the tibialis anticus, showing that some of the nerve-fibers in the external and internal popliteal nerves were still functioning. The peroneal group and the quadriceps group of muscles were completely paralyzed. All the extensors of the toes were active, particularly the extensor longus hallucis, which had taken on part of the function of the tibialis anticus.

We have had a number of these cases in the past few years and, therefore, have been obliged to do some real orthopedic surgery in order to give these patients useful limbs. We do not put mechanical supports on any of them after the muscles have contracted and the limbs are in a fixed position. This patient's foot was in extreme extension, and she walked on the outer edge of it—a talipes equinovarus. She had no power to dorsoflex it, because the tibialis anticus was paralyzed. You can see from the photographs what a good foot she has now. It strikes the ground flat and carries her weight well, and in place of turning in, it is turning out, that is, there is abduction instead of adduction.

We operated on her twice. At the first operation we split the tendo Achillis longitudinally for a distance of about two inches, and divided the outer portion, consisting of about two-thirds of the thickness of the tendon, transversely at the upper



Fig. 164.—Paralytic pes equinovarus. Old-standing peroneal paralysis the result of infantile anterior poliomyelitis. Before Dr. Murphy's tenoplasty on the Achilles and tibialis anticus tendons and cutting of the long plantar ligament. Lateral view of the foot.



Fig. 165.—Paralytic pes equinovarus. Old-standing peroneal paralysis the result of infantile anterior poliomyelitis. Before Dr. Murphy's tenoplasty on the Achilles and tibialis anticus tendons and cutting of the long plantar ligament. Internal view of the foot.



Fig. 166.—Paralytic pes equinovarus. Old-standing peroneal paralysis the result of infantile anterior poliomyelitis. Before Dr. Murphy's tenoplasty on the Achilles and tibialis anticus tendons and cutting of the long plantar ligament. Dorsal and inner view of the foot.



Fig. 167.—Paralytic pes equinovarus. Old-standing peroneal paralysis the result of infantile anterior poliomyelitis. Before Dr. Murphy's tenoplasty on the Achilles and tibialis anticus tendons and cutting of the long plantar ligament. Anteroposterior radiogram.

end of the split. The inner one-third was divided at the tubercle of the os calcis. The foot was then forcibly flexed, and the cut ends of the tendon were carefully approximated and sutured with a heavy chromicized catgut. The subdermal tissues were sutured over the reunited tendon with plain catgut and the skin with horsehair. That put the entire traction of the calf muscles on the outer two-thirds of the tendon and on the outer half of the tuberosity of the os calcis, instead of on the inner half, and also allowed the heel to come down to the ground.

Next the extensor longus hallucis was exposed at the ankle through a small incision and likewise over the head of the first metatarsal bone. It was freed and divided close to its attachment to the metatarsal bone. The distal end of the tendon was sutured to the side of the extensor brevis hallucis. The proximal end was drawn out through the ankle incision, passed beneath the skin, and attached to the fibrous tissue at the tuberosity of the fifth metatarsal bone, to bring the foot into eversion. The tendons were sutured with catgut. The skin wounds were closed with horsehair only.

Next the tibialis anticus was exposed and split into two parts longitudinally; one segment was divided transversely at its attachment to the scaphoid. The proximal end of this portion of the tendon was sutured into the transplanted tendon of the extensor hallucis, so as to get the double effect of both muscles as everters and dorsoflexors.

Then, with the small tenotome, we divided subcutaneously the plantar fascia attachment to the plantar tubercle of the os calcis. The fibulocalcaneal ligament was exposed by an incision over the external malleolus and shortened by reefing it. With the aid of a Jones wrench, which should be in every operating-room, the foot was manipulated until it was as flat as desired. A light plaster cast was applied with the foot flat and at right angles to the leg.

The patient made an uneventful recovery, and before she left the hospital she was fitted with a special shoe to support the foot in the proper position.

There remained, however, the complete paralysis of the quad-

riceps muscle group and of the sartorius—the latter is supplied from the same motor center in the cord as the quadriceps muscles, through the anterior crural nerve. The biceps femoris functioned normally, as well as all the muscles of the internal hamstring group. A second operation, therefore, was performed later, with the idea of giving the patient a useful knee by providing her with power of extension. She had no power to bring the leg into the extended position. The biceps, although functioning, was not well developed, but sufficiently so to be used for the purpose to which we intended to put it. The biceps tendon was freed from its attachment to the head of the fibula, drawn back for five inches, and then passed forward through the subcutaneous fat and transplanted into the quadriceps tendon at its outer side. In this way the biceps was converted from a flexor into an extensor muscle. She learned to use this muscle very well. If I had taken one of the muscles of the internal hamstring group, we should have had a much greater distance to span. We dressed the leg in extreme extension. The result was very satisfactory. She was able to use the leg and foot in walking very well, without mechanical support, when she left the hospital.

Our case of today differs from the one just cited only in the muscle groups involved in the paralysis. The history of this case is as follows:

HISTORY

The patient is a girl of eight years. She entered the hospital October 24, 1913, because of inability to extend her right leg and dorsoflex her right foot. Her present trouble dates back to July, 1910, when, during the night, she awoke crying because of pains in her head. Next morning she complained also of weakness in her legs, and her mother thought she looked feverish. A doctor was called who said she had fever, but ascribed it to an attack of indigestion. That night she complained of severe pains all over her body. The skin was extremely hypersensitive to touch. There was an epidemic of infantile paralysis in the neighborhood at that time, and three or four days following this illness her father noticed that her lower limbs were paralyzed,

a finding which was confirmed by the doctor. No other part of the body was paralyzed. She remained in bed four weeks, until the pain in her legs had subsided. After two months motion gradually began to return in the left leg and she was able to stand on that leg, but not on the right. At this time an osteopath was secured, who massaged the limb at regular intervals for one year. Six or seven months after the onset of the trouble a brace was applied, which was fastened to the shoe and extended to the middle of the thigh. She wore this brace until a year ago. Since then she has been walking with a cane. At present she can put some weight on her right leg.

Examination.—The quadriceps extensor is involved, because she cannot straighten her leg. The external popliteal nerve is also involved. She has function of the gastrocnemius. Plantar flexion at the ankle is perfect, showing that the internal popliteal nerve is intact.

COMMENTS AND OPERATION

DR. MURPHY: There was no question about the nature of this girl's illness. It was an anterior poliomyelitis, with a consequent paralysis of both legs, and a subsequent return of function in all of the muscles except certain groups in the right leg. The right adductor group of muscles is intact. The internal and external hamstring muscles, that is, the semitendinosus, semimembranosus, gracilis, and biceps femoris, are likewise intact; but the extensor quadriceps group of muscles is not functioning. She is unable to extend the leg or dorsoflex the foot, showing that there is likewise some involvement of the external popliteal. The internal popliteal appears to be functioning, because all the flexor muscles of the leg are intact; even the gastrocnemius muscle is functioning. She has perfect control of the rectal and vesical sphincters, and the anterior abdominal wall is strong, showing that the nerve supply of these regions has not been interfered with in the least.

Now what are we to do for this little girl? Remember, the internal hamstring muscles are intact, and the biceps femoris, constituting the external hamstring, is also intact. What this

little girl needs is power in her extensor femoris muscles. We must supply power to throw the leg forward, so that she can walk.

The plan we have decided to adopt in this case is the one we just described, namely, the transplantation of the tendon of the biceps muscle into the quadriceps tendon. We shall detach the biceps tendon from the head of the fibula, carry it forward under the skin and fat, and insert it into the middle of the quadriceps tendon, so that she will have ample power to extend her leg. This was the plan we adopted in the previous case, and it proved so effective there that we are encouraged to try it again. We have practically nothing to lose, because the internal hamstring group of muscles is sufficiently powerful to give this girl all the flexion she needs. If we can only counteract the ample flexor power by sufficient extensor power she will get, in the course of time, a very useful leg.

This case is typical of the whole group of cases. Unfortunately, in spite of all the study of anterior poliomyelitis, of its etiology, its symptoms, and its treatment, the fact still confronts us that the diagnosis in many of these cases is not made until after the completion of the paralysis. These little patients may not be ill enough to make it seem necessary to the parents to call in a physician until the arm or leg or both arms or both legs are powerless. When you stop to think that all this destruction of nervous tissue is accomplished inside of the first twenty-four hours, it makes you feel as though you want to throw up your hands in helplessness. And yet the present world-wide study which is being made of the affection may, and we hope it will, lead to something in the way of early diagnosis before the onset of the paralysis and before the serious involvement of the anterior horn cell-bodies. It is not sufficient to know the bacteriology of the disease. It is not sufficient to know that certain microorganisms and their toxins have a selective action on the cells in the anterior horns of the cord. What we must know, if we are going to do these patients the maximum of good, is how to prevent the destruction of these nervous tissues. Once the destruction of the tissue has occurred or even begun, treat-

ment will have little or no effect. Of course, prevention is a very effective means for combating the disease, and our knowledge of the transmission of the bacterial cause of the disease by the stable-fly, as asserted by Rosenau, may prove of tremendous value in lessening the number of cases; but we are still confronted by the fact that so long as we have acute anterior poliomyelitis with us, so long shall we have its sequelæ appearing in our clinics. The best we can do under present conditions is to give these people relief, to provide them naturally or artificially with what power of motion they need to enable them to walk and to use their hands to gain a livelihood, by tendon transplantation, arthrodesis, et cetera.

It is a well-known fact that in many cases of anterior poliomyelitis paralysis of much, if not of all, of the muscle function is subsequently regained. Why? Because the anterior horn cells in the cord have not been destroyed. They merely are involved in an inflammatory process not severe enough to cause necrobiosis, and the result is that their function is suspended, but not destroyed. As soon as the inflammation has subsided, the cells become active again, and the result is the disappearance of the paralysis. If the cells are dead, the paralysis is permanent. A return of function in paralyzed muscles always signifies that the motor nerve-cells from which are derived the axons of the nerve supplying the muscle have not been destroyed. Some of these cells may be destroyed without any appreciable effect on the function of the muscles. If many cells are destroyed, the muscle is weak. If all the cells are destroyed, the muscle is paralyzed, and the only thing you can do in such a case is to tap the power from some other nerve, either by neuroplasty or tenoplasty, and supply it to the paralyzed muscle. Sometimes function returns fairly rapidly in these paralyzed muscles. At other times a great deal of time elapses before there is any appreciable return of function. The extent and severity of the pathologic changes in the spinal cord around the involved cells determine the speed and degree of the return of function.

Often a doctor will tell his patient that there is nothing to be done for these paralyzed muscles. Such a statement is rarely true,

because if only certain cells are dead and function is merely suspended in the cells which remain alive, these latter cells, as we know, will in time supply the necessary function, or a large part of it, to the muscles.

Right here I want to point out to you a matter which has always been a greatly discussed subject with the profession. Why do these patients go to the quack, and why do they derive benefit from his treatment? They go to him because we have failed to do something for them. They always come to us first and they stand by us nobly, giving us plenty of time to help them. It is only when we fail that they turn to the quack. Now, why should not our patients get the benefit of the massage, the electricity, and the other mechanical measures with which the quack salves them? They get some return of function in their paralyzed limbs! They get a result! That is all they want. And why do they get it? Because the function in their anterior horn cells was merely suspended by the inflammation and edema compressing them. We all should know that in the course of time many of these cells will regain their function and that time will tell the story. You should make it clear to the patient that a definite, well-recognized improvement is going to take place and explain the reason why. Naturally, when they get well after having visited the quack they give him the credit for the improvement. That is the point I want to impress upon you. When these cells are dead, they are dead for all time; but when their function is merely suspended, it is bound to return in the course of time. Thus we see why some paralyses are permanent and others merely temporary. In the early management of these cases it is important to understand these facts fully.

How long does the improvement in motion continue? About two and one-half years. At the end of that time the limit of function has been reached in most cases. You can always test out these nerves with the faradic current, because if there is merely a suspension of function in the nerves, if the axons are alive, the faradic current will induce a response in the muscles supplied by them. If the nerves are dead, that is, if their cell-bodies are dead, the faradic current will not induce a response

in the muscles. The galvanic current, on the other hand, arouses muscle action in a muscle which is deprived of its central nervous connection. Therefore it is essential to make a careful electric study of every case of paralysis for the purpose of determining which nerves are alive and which are not. If you will reëxamine the patient from time to time, determining the faradic reaction, you will be able to make a fairly accurate estimate as to how the restoration of function is progressing and how much more improvement you may expect. You owe that attention to your patient.

Now, as to the case before us. The technic of the operation which we shall do is very simple—in fact, tendon transplantation as a whole is rather an easy procedure. The reproduction of tendons where they have been destroyed is not such a simple matter. Tendons are notably hard-lived tissues. You can do a great deal to a tendon and still have it live. They live by osmosis rather than by vascularization; therefore you need not worry particularly about their blood-supply. If you have not tendon enough for your purpose, you may lengthen it by splitting it or you may make a new tendon by using some fascia and fat, as, for instance, a portion of the fascia lata. Roll it or twist it and make the tendon as long as you please. We did that some years ago at the Presbyterian Hospital for a man who had the tendons on the anterior surface of his wrist destroyed. We made a pedicled skin, fat, and fascia transplant from the abdomen, to cover the defect in the wrist and hand. Through this healed transplant we tunneled for our tendon transplantation and secured a splendid result. The tendon was made by rolling a strip of fascia lata and fat.

In this case we make the incision just in front of the external hamstring, so as to expose the biceps tendon and the quadriceps through the same incision. We are very careful in operating in this field because of the nearness to the external popliteal nerve, which we do not wish to traumatize or injure. Remember that it winds around the head of the fibula from behind forward, and that the biceps tendon is attached to the outer posterior surface of the head of the fibula in close proximity to the external pop-

liteal nerve. Retracting the edges of the incision, the tendon of the biceps is easily exposed. There it is, and just a little lower down, you see, is exposed the external popliteal nerve. We shall free this biceps tendon from its attachment to the head of the fibula, going well down on the surface of the fibula. Remember, the tendon passes down over this surface as a sheath. A hemostat is attached to the proximal end of the tendon, so that we shall not lose it in our work.

Next I shall expose the quadriceps extensor tendon and then split the biceps tendon into halves, pass the whole tendon through a slit in the middle of the quadriceps tendon, and then wrap the split halves around the quadriceps, one-half being carried to either side and attached securely by means of chromicized catgut sutures to the quadriceps. That gives a secure fixation. The biceps cannot possibly get away from the quadriceps.

From now on this patient will have to train herself to use this flexor muscle as an extensor. From our previous results in these cases we feel confident that she will be able to do this. The leg will be dressed in extension until the tendons have become fairly well united, say five or six weeks, and then the patient will be encouraged to use the limb. We are always careful of the external popliteal nerve in all this transplantation work and, in fact, in any work, in this vicinity, because any injury to it will result in a drop-foot, a condition which one sees frequently in fractures near the knee-joint, and sometimes in fractures even at some distance from the knee-joint, owing to pressure on the external popliteal nerve from a splint, bandage, cast, or other retention apparatus. There is no good reason why the nerve ever should be traumatized in operating, because it is always either clearly in view or easily palpable, even through the skin. Like the ulnar nerve, you can always find it and you should always look for it to protect it against trauma.

In connection with our present case I should like to call your attention to a few foot cases which required somewhat similar procedures.

The first case was an exceedingly interesting one, first, because of the extensive reconstructive work which was done; and,



Fig. 168.—Pes equinovarus from peroneal paralysis, the result of anterior poliomyelitis. Before the operation. Foot firmly fixed in this malposition without power of dorsal flexion.



Fig. 169.—Same patient after lengthening the Achilles tendon and transplanting it to the outer tubercle of the os calcis, and transplanting the tibialis anticus tendon to the base of the fifth metatarsal. Plantar fascia cut at attachment to plantar tubercle of os calcis and intertarsal plantar ligaments cut. Redressment by Jones' wrench. Photograph shows the degree of extension possible after operation.



Fig. 170.—The same patient. Extent of flexion possible after operation.

second, because of the effectiveness of the result obtained. The patient, a boy aged fifteen, was admitted to the hospital February 2, 1912. The family and personal history was negative. The patient never had had any injury or rickets or other bone disease—nothing that could have any relation to the trouble for which he entered the hospital; no chills, fever, or infection.

When seven years old his mother noticed that he wore through the sole of his left shoe more quickly on the outer side, near the front of the foot, than elsewhere, but she could see no deformity of the foot. He played like other children. Not until three years later, in 1907, was a deformity of the foot noticeable. Since that time the deformity has been increasing in degree. There is no pain except a dull ache after standing on the foot for some time.

Examination.—There was no tendency of the foot to turn either in or out. The Achilles tendon was strongly retracted, but the heel touched the floor, under great strain, however, when the patient stood erect; but when in a resting position, he had a marked equinus with the articular surface of the tibia resting on the posterior surface of the astragalus. Not only equinus, but also a tendency to eversion of the ankle, was present. There was much contraction in the plantar fascia, which was very short and formed an extremely high arch; no paralysis in any muscles of the anterior tibial, peroneal flexor, or extensor groups; no evidence of rickets or any other bone disease. Heart and lungs were normal. The urine was negative.

What did we do for him? First we lengthened his Achilles tendon. The inner half of the attachment to the os calcis was taken off with its periosteum. The Achilles tendon was then split upward a distance of two and a half inches, and its outer half cut through at that point. This procedure permitted of extension of the foot to the full degree. After rupturing the posterior annular ligament with repeated extension efforts so that the foot could readily be put in and easily held at an acute angle to the tibia, the ends of the divided Achilles tendon were reunited, transferring the full traction of the tendo Achillis to the outer tuberosity of the os calcis, making an elongation of about three

inches in the tendon. The future traction of the Achilles tendon must all come on the outer half of the tuberosity of the os calcis, and there is no force acting on the inner half. The plantar fascia was then divided subcutaneously just anterior to its attachment to the plantar tubercle of the os calcis, and the plantar, tarsal, and metatarsal ligaments were divided clear across the arch. This procedure made possible a fair reduction of the pes cavus. The external half of the tendon of the tibialis anticus was split from its attachment to the internal cuneiform bone and reflected upward as far as the annular ligament. The periosteum and ligaments were then exposed on the upper surface of the cubometatarsal articulation. A hemostat was passed beneath the tissues to the opening at the end of the annular ligament, and half of the tendon of the tibialis anticus was grasped and drawn out and sutured to the periosteum of the cuboid bone at its outer and anterior portion. Sufficient tension was put on this junction to produce pronounced relaxation of the internal half of the tibialis anticus, which was left attached to the internal cuneiform bone. The purpose of this reattachment was to convert the tibialis anticus from an internal rotator of the foot into an external rotator, and to balance the contractor factors of the foot so that the astragalus would be held plumb beneath the articular surface of the tibia.

All the plantar flexor tendons of the toes were divided over the middle of the first phalangeal bones without suture. By the transposition of the attachment of the Achilles tendon to the outer side of the tuberosity of the os calcis, and of the tibialis anticus to the cuboid, the foot was drawn outward at every muscular contraction, either in extension or flexion. This was beautifully illustrated when the boy was examined four weeks after the operation. With every effort to extend or flex the foot it was abducted instead of adducted, and he walked plumb on his astragalus.

I feel that this correction of the pes cavus should never be performed by the heavy crushing instruments in use by orthopedists up to this present time. They are unsurgical as well as unnecessary. When the tibialis anticus is out of commission,

the extensor longus hallucis or extensor communis digitorum or even the peroneus brevis may be effectively used to assume the function of the tibialis anticus. The same method of elongation should be carried out in the congenital deformities of the upper extremity and the same surgical principles applied.

Another somewhat similar case was the following:

The patient, a girl aged fifteen, entered the hospital October 28, 1913, because of deformity of the left foot, with very limited motion in the left ankle. Her present trouble dated back to thirteen years ago (1900). The patient at that time became suddenly ill, with a high temperature, which was associated with pain and hypersensitiveness all over her body. The next day there was noticed a paralysis of the left leg and right arm. The paralysis in the arm lasted only one day. The patient remained in bed for a long time, and when she did get up, she had to walk with the aid of crutches and a cane. Her parents state that there was no epidemic of infantile paralysis in the neighborhood at that time. A year later she was taken to a hospital, where an operation was performed on the left foot. The tibialis anticus was split and transplanted. Since the operation she has been wearing a brace continuously, extending from the foot to the middle of the leg. No other part of the body remained paralyzed.

That is a typical history for a case of this kind. This girl undoubtedly had an anterior poliomyelitis, followed by paralysis within twenty-four hours. Her final trouble was with her foot. She had a paralysis of all the extensors of the foot. The internal popliteal was the nerve primarily involved, because that is the nerve which supplies the muscles which extend the foot. She also had an inversion of the foot, besides being unable to extend it; but the dorsal flexors of the foot were intact.

At the operation which we did in this case we transplanted the tibialis anticus tendon to the outer side of the foot. That proved to be a rather difficult procedure, because of the earlier operation which had been performed on this tendon. Our plan was to convert that muscle from an internal to an external flexor, so that the foot would be plumb with the leg. Ordinarily we only transplant a portion of this tendon, but in this case it was

necessary to transplant the entire tendon to the outer side of the foot.

The tendon was exposed by an incision on the back of the foot, midway between the two malleoli. A careful dissection had to be made because of the large amount of scar tissue in which most of the tendons were involved. The tendon of the tibialis anticus muscle was located and freed very carefully from its surrounding adhesions, and when we had it ready for transplanting, a second incision was made directly over the base of the fifth metatarsal bone. The skin between the two incisions was undermined, and the tibialis anticus tendon was drawn through this tunnel and sutured securely with chromicized catgut to fibrous tissue and periosteum surrounding the head of the fifth metatarsal bone. The distal part of the tendon was tacked to the tendon of the extensor hallucis longus. The tibialis anticus was thus converted into an elevator of the outer border of the foot instead of an elevator of the inner border. Thus we made it possible for the foot to strike the ground flat and also made it possible for the patient to walk with more ease. The extensor tendon of the great toe was sutured to the side of the distal portion of the tibialis anticus tendon merely to aid in elevating the foot.

That was all that we did at that operation. The patient was advised to return at a later time for a second operation, to be performed for the purpose of giving extensor power to the foot. We intended to transplant some of the long dorsal flexors into the Achilles tendon. However, the patient has not returned up to the present time. She will probably do so in the course of the next few months. The result of our first operation was good. The foot was kept in a cast for several weeks, and when the cast was finally left off, an adhesive plaster toe elevator was applied for a few weeks. Then the patient was encouraged to walk, which she did fairly well and with gradual improvement.

Another case of the same class was the following:

The patient, a boy, aged ten years, entered the hospital January 26, 1914, with the following history. When he was four years of age (*i. e.*, in 1908) his parents noticed that he limped on

the left foot. Previously he always had walked without limping and had no deformity of foot or leg. He limped more and more as time went on, and the toes of his left foot gradually turned inward. When questioned at that time by his parents as to the cause of his limping, the patient replied that about two months previously he had struck his foot (in the region of the external malleolus) on a chair. There had been no physical incapacity at that time, however, nor could his parents find any trace of a

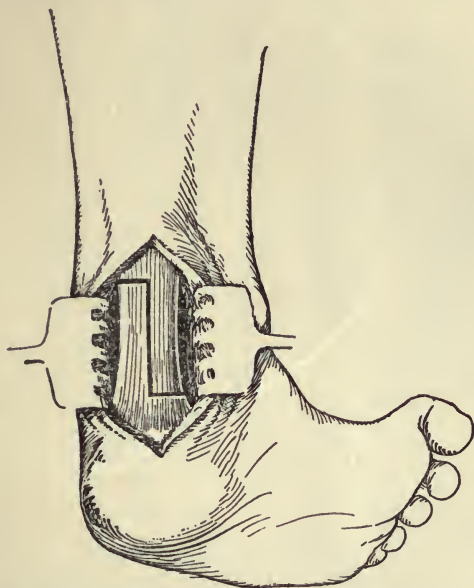


Fig. 171.—Achilles tenoplasty in paralytic pes equinovarus. Open elongation of the tendo Achillis and transference of its action to the outer tubercle of the os calcis (see text).

First Step: Incision in the tendon.

bruise to substantiate this story. They also noticed that the left foot, leg, and thigh were not developing in size so much as the right limb. He had a typical talipes equinus walk, but never any pain.

There was nothing out of the ordinary about this case. At the same time let us analyze it and see what happened. In talipes equinovarus you have, first, a contraction of the plantar

fascia; second, a contracture of the tibialis anticus; third, when the patient walks there is a relaxation of the peroneal group of muscles, which are attached to the outer side of the leg. As a

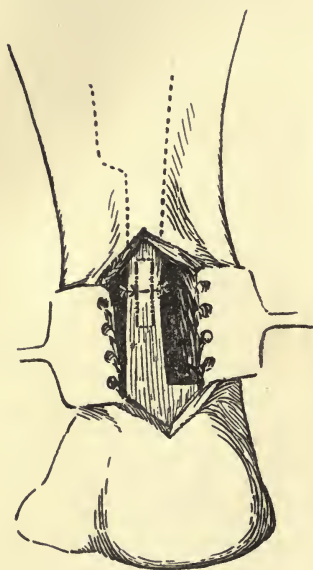


Fig. 172.—Open elongation of the tendo Achillis and transference of its action to the outer tubercle of the os calcis.

Second Step: Lengthening of the tendon. Uniting the two cut ends by suture and thus transferring the pull of the calf muscles to the outer side of the axis of the ankle.



Fig. 173.—Achilles tenoplasty in paralytic pes equinovarus. Subcutaneous tenotomies for the elongation of the tendo Achillis and transference of its action to the outer tubercle of the os calcis (see text).

First Step: Incisions and method of making the tenotomies. Both the lateral incisions in the tendon are represented as made (indicated by dotted lines), while the tenotome is depicted in the act of making the longitudinal incision in the tendon.

result of the firm contracture of the tibialis anticus there is a bowing outward of the arch of the foot and a shortening of it. After the condition has existed for a time, a third element comes

into play,—a shortening of the tendo Achillis,—so that there is a deviation of the center of gravity from the middle of the articular surface of the astragalus.

How are we to overcome the condition? By the same de-



Fig. 174.—Achilles tenoplasty in paralytic pes equinovarus. Subcutaneous tenotomies for the elongation of the tendo Achillis and transference of its action to the outer tubercle of the os calcis.

Second Step: Cut ends of tendons brought together by forcibly dorsoflexing the foot and stretching the tendon.



Fig. 175.—Tenoplasty for pes equinovarus. Method of splitting the tibialis anticus tendon from its attachment at the base of the first metatarsal backward to the ankle and transplanting its outer half to the base of the fifth metatarsal bone to correct inversion by dorsoflexing the outer side of the foot.

tailed procedures as in the previous case. First, we cut the tendo Achillis, taking off two-thirds of the tendon from its attachment to the inner side of the os calcis, split it upward, and then on out to the outer third. Elongate the tendon by uniting the

inner two-thirds to the outer third. That puts all the lower attachment on the external third of the tuberosity of the os calcis.

Second, expose and divide or split the tibialis anticus tendon. When there is a high degree of the deformity, we transplant the whole tibialis anticus tendon to the outer side of the foot. If the deformity is not so marked, we transplant only half of the tendon.

Third, the shortening of the plantar fascia must be overcome. You will remember how Phelps used to cut across all the plantar structures, right down to the bone, with the hope that later they would reunite in better position. Sometimes they did unite and sometimes not. Sometimes they united very expensively for the patient. The plantar fascia is attached behind at only one spot, the plantar tubercle of the os calcis. In front it spreads out like a fan. Do you need to cut it in the middle? Do you need to cut it where all the vessels and tendons lie? No. All you need to do is to detach it subcutaneously where there are no nerves, no tendons, no vessels to cut. Divide it, as in the case already cited, just anterior to its attachment to the plantar tubercle of the os calcis. Then, after dividing the intertarsal plantar ligaments subcutaneously, use the Jones wrench to swing the foot around. That disposes of the three pathologic elements involved. You need not take out a portion of the bone at all, nor do you injure anything important. You cut the plantar tendon like the string of a bow. That is all.

By the preceding procedures we not only straightened the foot, but also overcorrected it, since merely to straighten it is not sufficient.

Another case belonging to the same group was a woman with a double drop-foot. We did a subcutaneous tenotomy on the tendo Achillis in both feet, and divided and transplanted both tibialis anticus tendons as in the preceding case. We also divided her plantar flexor longus hallucis. This patient regained splendid power of extension in her feet, but very feeble flexion. She had no paralysis of the quadriceps.

There is a wide and fertile field for tendon transplantation. It is astonishing how much can be done to restore lost muscle function merely by transplanting one or more tendons, even from

an opposing muscle group. However, great selective judgment must be exercised if one is to be successful in this work. Otherwise, the wrong tendon may be transplanted to the right place or the right tendon to the wrong place, or, less frequently, both errors may be made simultaneously. The results obtained in this work are exceedingly gratifying. We have had a considerable number of these cases, and while we have not always been able to restore all the lost function, we have succeeded in giving these patients useful limbs.

The history of the last leg case we shall cite in this connection brings out well a point which we have emphasized frequently in this clinic, namely, *avoid a tight cast*, especially on the leg, because the pressure of the cast on the external popliteal nerve, where it winds around the head of the fibula, may suspend this nerve's function and cause foot-drop. That is exactly what happened in this case. Following an injury, a plaster cast was applied by a country surgeon to the leg and was not cut. There was neither a longitudinal splitting of the cast, as is the custom here in our clinic, nor was a window cut out over the external popliteal nerve, which is our other alternative to avoid compression of this nerve.

The patient was a man aged twenty-three. He entered the hospital on April 4, 1913, seeking relief for a deformity of his right foot. His foot would turn in when walking, so that he put his toes to the ground first. Then he had to bend the knee and lift and swing the foot outward and forward at each step in walking. He cannot put the heel on the floor without advancing the foot well out in front of him.

His trouble dated back to April 18, 1911, when, on attempting to mount a horse, the horse ran away and jammed the patient's right leg against a post. He was thrown to the ground by the impact. On attempting to rise he noticed a false point of motion about four inches above the right ankle. He had pain at the site of this injury and great tenderness. The bones did not protrude through the skin. The leg was swollen.

The fracture was reduced and the leg put in splints and bandages for two months. He had pain only for the first week

or so. At the end of two months the splints were removed and it was found that the union was not good. A circular plaster cast was then applied, extending from the toes to above the knee; but it was not cut out over the external popliteal nerve. It was taken off at the end of three weeks. There was some deformity, but good union. He used crutches and then a cane. He had no pain. About four months after the accident he was able to walk without a cane, but the leg was shorter and he thinks that the foot was inverted a little at this time.

October 5, 1911, he had another operation for the shortening and Lane plates were put in. A circular cast extending from the toes to the hip, but not pressing on the external popliteal nerve, was applied, with a window cut over the wound to permit of dressing. The cast was removed in two months: the union was good, but the foot was inverted.

The patient now is not able to evert the foot. He can flex all his toes except the fourth and fifth.

The indications for our treatment in this case were clear. What the man wanted was power to throw his foot forward and outward, that is, to dorsoflex it. This was not a case of Pott's fracture, according to the history, and I believe the history is correct. If this man did have a Pott's fracture, he had a result which I never before have seen follow a Pott's fracture. The Pott's is an eversion fracture. The foot is thrown outward and not inward, as it was in this case. This man's tendo Achillis was short, and he had the characteristic deformity and gait which go with a short Achilles tendon; that is, he walked on the outer side of his foot. Therefore, to correct the deformity, we lengthened his tendo Achillis and transplanted his tibialis anticus, as was done in the previous cases. In dividing the tendo Achillis in these cases we leave only the outer third of it attached to the os calcis, the inner two-thirds of the insertion being separated from the os calcis, the tendon split upward and the inner third of the proximal portion reunited to the outer third of the distal portion so that the pull on the heel is from the outer side instead of from either the entire Achilles attachment or the inner side of the attachment.

The division of the Achilles tendon in this case was accomplished subcutaneously. The foot was put in extreme flexion to make the tendon tense. Three small skin punctures were the only wounds made; the inner half of the tendon was cut from the os calcis through a skin puncture; the outer half was divided two inches above the os calcis, through a similar puncture; the tendon was then split subcutaneously by joining longitudinally the inner ends of the two transverse incisions. When the foot was forcibly flexed, the two halves slid by each other until the ends rested in contact, and there the foot was dressed. It was also necessary to incise the posterior annular ligament in order to bring the foot around as far as it should come. That, too, was done subcutaneously. If one keeps in mind the anatomy of the ankle-joint, a subcutaneous division of these tendons and ligaments may be done without danger to the vessels or nerves in the vicinity of the joint.

TENOPLASTY ON WRIST; ADHESIONS OF ALL TENDONS EN MASSE; FREEING AND WRAPPING OF THE SUPERFICIAL GROUP IN A FAT AND FASCIA FLAP

HISTORY

THIS patient, a man thirty years old, entered the hospital December 15, 1913, because of partial inability to use his left hand and fingers. January 18, 1913, his left wrist, thumb, and first finger were caught in a machine, breaking the radius about two inches above the styloid process. The broken ends of the bone did not project into the wound, but the wrist was deeply cut on its flexor surface, everything being severed to the bone. The distal phalanges of the thumb and first finger were crushed. They were amputated when the tendons were sutured. A splint was applied.

He was in bed for one week, had no chills, but had a slight fever for three days. The wrist wound healed after six weeks; the fingers, after eight weeks. The splint was discarded about six weeks after the accident. Then the patient found that his hand and fingers were stiff and slightly flexed. October 2, 1913, he was operated elsewhere in the hope of freeing the tendons.

Examination on December 16, 1913, showed that the ulnar and median nerves evidently had been cut, because sensation was absent from the palmar surface of the hand; the carpal tendons were agglutinated and the radial pulse absent. He had slight flexion of the left hand at the wrist, but no flexion of the fingers.

COMMENTS AND OPERATION

DR. MURPHY (December 17, 1913): What this man wants is to regain the use of his fingers. Now he has no finger action at

all. His flexor tendons appear to be fixed and matted together, so that when he attempts to flex his fingers his hand moves only at the wrist. So far as we can learn he had no suppuration in his wrist following the injury. From the fact that he now has an atrophy of all the muscles of the hand and no return of muscle function, we are of the opinion that the median and ulnar nerves, which were found divided at the operation, in October, were not reunited. The radius fracture is healed, and the flexor tendons have become fixed to the callus. It would be useless to take out that portion of the radius, because of the matting together of the tendons. Cases like this are very unpleasant, because the extensive pathologic lesions present render impossible an ideal result. We shall do the best we can for him, however, and try to give him some finger flexion. Just what our procedure will consist in we cannot say until we have exposed these tendons.

This exposure we shall make through a longitudinal incision on the anterior surface of the wrist, excising the old scar tissue as we enter. The tendons are so matted together that I cannot recognize a single tendon, nor can I tell which strand is tendon and which is nerve in this mass of connective tissue. Therefore, we shall have to elongate the incision upward in order to bring some of the muscles into view; then we shall be able to identify the tendons. Here is a portion of old catgut suture in this connective-tissue mass. It is an unabsorbed remnant from the previous operation, and will be removed.

The first structure we want to locate now is the median nerve. By a blunt scissors dissection we find here something which looks like a nerve. Let us apply the faradic current. If it is a nerve and alive, we shall get a response. If it is a nerve, but dead, we shall get no response. Testing the nerve in this way, you see there is a response, thus showing that the axons in the nerve are alive and capable of transmitting impulses; but from the fact that the normal impulses are not transmitted we are convinced that the nerve has been compressed by the cicatricial tissue in which it is embedded. Therefore, we shall free the nerve very carefully throughout its entire length from this connective tissue, and shall make provision before we finish the

operation that this nerve will not again become constricted by the formation of new cicatricial tissue.

Now let us look for the ulnar nerve. That is a much more difficult search. We shall begin the dissection higher up in the arm, because higher up we can locate the nerve readily. Then we shall simply follow it on down into this connective-tissue mass from which we expect to liberate it. See! This nerve has been divided and not reunited. On its proximal end is a neuroma. Its distal end, which we find is separated from the proximal by about three-fourths of an inch, also has a small neuroma on it. I think we shall be able to reunite this nerve without very great tension. Even if we fail to get an accurate approximation of the two ends, we shall get union if only we can succeed in keeping out interposing tissue. Levings, of Milwaukee, some years ago showed us that the axons of nerves often will span a considerable distance through muscle if directed parallel to the muscle-fibers. The chemotropism of Thoma attracts the divided axons to one another.

Now that we have restored the continuity of the nerves, let us return to the tendons. The radial artery was ligated at the original operation in October. The proximal end of it can be seen very nicely here. It is a hopeless task, I think, to try to separate these tendons from this large mass of scar tissue. They would certainly adhere together again subsequently. I think we have done very well to reunite the cut ends of the ulnar nerve because that is the important nerve for this hand. If we can restore its function and give this man some power in his fingers, he should be satisfied. The patient is a locomotive engineer. If he can again hold the throttle lever, he will be able to work. That is really all he asked of us, to give him that much finger power. These tendons at present are all bound down in one solid piece. We shall free the superficial group of tendons *en masse* from the deeper group (including the carpal flexors and the flexor sublimis), so that when he attempts to flex his fingers each group will all move as one tendon. He will not be able to flex his fingers separately, but he will flex them all together and with sufficient strength for a good grip.

In order to prevent a reunion of the superficial group of tendons to the deeper group, I shall wrap the former in a flap of fat and fascia which I shall remove from his fascia lata. I shall take a piece about three inches in length and two inches in width, wrap it around the tendons and suture it in place, so as to form a bed for them. If we can make this transplantation without getting the flap infected, the latter will live and our object will be attained.

This free transplantation of tissue flaps (Lexer) is not new. Such flaps have been used to fill cavities in bone, and defects after tumor removal, as, for instance, after excision of the breast. Here, for cosmetic reasons, the contour of the chest may be restored by transplanting a flap of fat and fascia. Similar flaps have been used to prevent adhesion of skin to exposed bone, as, for instance, in operations on the face. Likewise new tendons may be made from these fascial flaps. They also make admirable material to prevent the occurrence of adhesions around tendons either individually or *en masse*. We usually use egg membrane to wrap around the point of union when we are dealing with tendons individually. Egg membrane is easily obtained and can be made absolutely aseptic. It answers the purpose about as well as the transplanted tissue. We shall not do anything with the median nerve, except give it a new bed to free it from further constriction by the scar tissue. The skin is closed in the usual manner.

This hand was a much more difficult proposition than the average case, because of the extensive pathologic changes present. The hand was absolutely useless to this patient in the condition it was in. Something had to be done for him. I think we have succeeded in our efforts and I feel very hopeful of the result. We shall dress the hand in an elevated and slightly flexed position with a posterior splint of wire-gauze. [*Note.*—Healing *per primam*.—ED.]

TRAUMATIC DIVISION OF FLEXOR TENDONS AND MEDIAN NERVE; TENOPLASTY AND NEUROPLASTY

HISTORY

THIS patient is a married woman, aged thirty-one. She entered the hospital February 13, 1914, with the following history: On February 19, 1912, while descending a stair, she slipped. Throwing out her right arm to protect herself, she pushed her hand through a window-pane and cut a deep gash across the anterior surface of the wrist. The doctor who was called told her that the tendons of the thumb and index-fingers were severed, but that the nerves and arteries were intact. He reunited the severed tendons and closed the wound without a drain. It was dressed for four weeks. At the end of that time she was told to use the hand. She says that the palmar surface of the hand, including the thumb and index- and ring-fingers, looked shrunken then and has remained shrunken. The hand was hypersensitive over this area, and also on the dorsal surface of the second and third phalanges of the index- and middle fingers. These areas are hypersensitive still. She is unable to flex the thumb, but can extend it. She can flex the index-finger only at its metacarpophalangeal articulation. She flexes the middle finger at all joints, although slowly and incompletely. Her grip is poor.

COMMENTS AND OPERATION

DR. MURPHY (February 14, 1914): The findings in this case make it clear that something is the matter with the flexor tendons. The patient can extend all her fingers, including the thumb; but she cannot flex the thumb and she can flex only partially the index-finger. The power to flex the phalanges of the index-finger is absent, that is, the flexor indicis is out of function. The

flexor sublimis also is not acting nor is the flexor carpi radialis. The flexor profundus is not so strong as it should be. These facts suggest that there may be something wrong with the median nerve which supplies these muscles. The flexor carpi radialis and the flexor sublimis digitorum are supplied by the median nerve. The muscles supplied by the ulnar nerve flex the hand and the first and second phalanges of the fingers and they appear to be active. The profound flexor, however, which flexes only the third phalanges and is supplied by the median and ulnar, is functioning only partially. How the patient could have her flexor profundus cut by the window-pane without also cutting the sublimis one cannot understand, because the former rests underneath the latter. Sensation on the inner side of the ring-finger and on both sides of the little finger is normal, but there is hypersensitiveness in the other fingers. Remember that this woman was cut by glass. A glass cut is not like a razor cut. There is no uniformity to the cut and you cannot tell which structures will be cut and which spared. Often glass will cut only on one side of a transfixed structure.

This woman has been operated once before, shortly after the injury occurred, and we are told that the tendons were sutured at that time. Perhaps only the sublimis tendons were sutured and the profundus tendons left unsutured. If these latter tendons were cut and then sutured, why is their function now suspended? It must be that the tendons either were cut and not sutured at all, or that they have become immobilized by union to the structures in the neighborhood. A third possibility is that there is something wrong with the nerve-supply. It is quite possible that the median nerve was divided and not reunited.

We shall make a longitudinal incision on the anterior surface of the wrist, so as to expose the anterior surface of the carpus. We shall have to be very careful in making the dissection because we do not know just how the anatomic relations may be disturbed. Retracting the wound edges, the first structure coming into view is the median nerve. You can always distinguish a nerve from a tendon by its pale pink color, whereas tendons are

white and glistening. We shall free this nerve from its bed. There should be a neuroma on both ends of this nerve if it was divided and not properly reunited, and there *is* a neuroma here, but only on the proximal end of the nerve. The distal structure to which this nerve was united has not the appearance of a nerve at all. Lifting it up very carefully and making tension on it, we find that it flexes the index-finger. That clears up the situation very nicely. See what has happened! The proximal end of the divided median nerve has been united to the distal end of the long flexor of the index. The nerve supply from the median, you see, has been cut off right at this point and all the distal structures supplied by it have been without a nerve-supply ever since the accident. That finding accounts for the absence of so much of the flexor power of these fingers. Now we shall try to find the distal end of this median nerve. There it is, drawn way over to the ulnar side of the hand and embedded in connective tissue. It has not become united to any of the other structures, and there is a small neuroma on its end. We must determine whether this distal portion of the nerve is alive, that is, whether its axons are capable of transmitting impulses. When we apply the faradic current we find that there is no contraction of the distal muscles supplied by this nerve. We have every reason to believe now that when these two segments of the nerve are reunited properly, there will be, in the course of time, say, about eighteen months, a complete return of function. At least such has been our observation in other cases of neuroplasty. We shall not reunite this nerve just yet, but shall continue our dissection of the tendons.

The flexor pollicis evidently has been divided, likewise the flexor indicis; but neither of these tendons has been properly reunited—in fact, the only tendon sutured was the distal portion of the flexor indicis, which was united with the proximal portion of the median nerve! The ends of these tendons have retracted considerably so that it is next to impossible to reunite them as intimately as we should like. However, we can make a fairly good approximation of the flexor indicis, and we shall resort to transplantation of some tendon to restore the function of the

flexor pollicis, the ends of which are so widely separated that they cannot be approximated. The only tendon which we can spare here for transplantation is the palmaris longus; but we can make use of the flexor carpi radialis to restore the flexor power in the thumb and depend on the flexors of the fingers and ulnaris to flex the wrist. We now have the divided ends of these tendons well straightened out, so that we know exactly what we have to do. We shall unite the distal end of the flexor longus pollicis to the proximal end of the flexor carpi radialis and we shall free the palmaris longus from its attachment and suture it where the carpal flexor was attached, thus making a carpal instead of a palmar flexor of it.

Now we are ready to approximate the cut ends of the median nerve. We must cut off the neuroma on both ends and we must make sure that the axons are in contact. Otherwise union of the axons will fail to take place, and there will be no transmission of impulses. We have exactly the same condition here as in the case of an ununited fracture of a bone. So long as the ends of the fragments are covered by fibrous capsule, soft tissue, or any other interposed material, union cannot take place. So long as the ends of the divided nerve are covered by any interposing material which prevents axonal contact, the function of the nerve cannot be restored. Therefore, we remove all this interposing tissue from the distal and proximal ends of the divided nerve, so that the axons fairly stick out, almost like a bristle-brush. Now we know that we can get a proper union and are sure that function will be restored.

That step completes this portion of the work. The former surgeon's result in this case was perfectly pardonable, because the operation was done in an emergency and not under the best conditions for aseptic surgery. We have been able to restore the normal conditions fairly well. In substituting tendons, as we did in this case by making the flexor carpi radialis take on the function of the thumb flexor and transplanting the palmaris longus to take on the function of the flexor carpi radialis, one must always keep in mind that *it is essential not so much to restore all the lost functions as to restore the important functions.*

It is certainly far more important for this woman to be able to flex her thumb than to flex her hand toward the radial side. We have not interfered with the flexor carpi ulnaris, and this latter muscle, together with the finger flexors, will give her as much hand flexion as she really needs. She must have thumb flexion, however, because otherwise the hand would be practically useless.

These tendons are short; but our first aim is to get them properly united; then, if necessary, we can operate again and elongate the tendons to overcome the shortening. The nerve function will return very slowly. Our experience has been that usually about eighteen months elapse before there is any considerable return of function. It does not matter how long after the nerve has been divided that approximation is made, because if you get an accurate approximation of the axons, or what we term an "axonal contacting," union will take place and function will return.

In closing this wound we must protect the sutured tendons, so that adhesions will not form to them and nullify our work. Therefore, we put each tendon into a separate connective-tissue bed, and in closing the skin we turn the cut edges outward, making an ectropion just as we do in closing the peritoneum. That will prevent fixation of the skin to the tendons. We dress this hand in the position which will put the least possible tension on the tendons and nerves. We shall let it fall into place. You see how the thumb is flexed fairly well into the palm of the hand and how the index-finger comes down over the flexed thumb. The other flexors have not been disturbed. We have closed the incision in the skin in the usual manner and applied the usual dressing. We shall now put on a posterior wire-gauze splint which will keep the hand flexed. We find these wire-gauze splints very useful and they are easily applicable to any case. They are light and not the least bit cumbersome. Where they can be used, we prefer them very much to plaster casts.

BONY ANKYLOSIS BETWEEN ULNA AND HUMERUS FOLLOWING FRACTURE OF OLECRANON; ARTHROPLASTY

HISTORY

THE patient, a man aged forty-eight, entered the hospital January 23, 1914, with the following history: On February 8, 1913, he was thrown off his feet. In falling he struck on his right palm, his arm being in full extension. The arm twisted and gave way at the elbow. There was immediate sharp pain in the elbow, inability to use the arm, and a swelling appeared about the elbow within half an hour. A radiogram was made and showed a fracture of the base of the olecranon. The fracture was reduced without anesthesia, and a straight splint, holding the arm in full extension, was applied. This splint was removed at the end of three weeks. There was then no motion in the elbow. Under ether anesthesia the adhesions were broken up. The elbow remained very painful, but without motion. Since March, 1913, the patient has tried various means to restore motion and relieve the pain. Toward the end of July, 1913, the pain commenced to subside, without obvious reason, but there was no return of motion. He does not remember having had a chill at the time of the accident; but he thinks that he had some fever about four days after the accident, and that the fever continued for about three days.

There is now present some tingling in the fingers and hand; he does not remember when this was first noticed. The thumb and first finger are constantly numb. The grip of the first and middle fingers is much weaker in the right than in the left hand. The finer sense of touch is impaired in the right hand. Pronation and supination are present to normal degree.

COMMENTS AND OPERATION

DR. MURPHY (January 24, 1914): I want to bring out a fact which is not mentioned in the history, and that is: Was the splint taken off at all during the first three weeks? In other words, could there have been a splint compression causing an ischemic myositis? The splint *was* removed. What then can have caused his ankylosis? In the management of fractures near joints we are constantly stating that forced, painful, passive motion should not be resorted to before the end of the fourth or fifth week, just the opposite to what we taught formerly, when we started giving passive motion from the first week. Let us mentally bring out that fracture from beneath the skin into the open. Suppose that you had a fracture of a bone, and after the fourth or fifth day you began pulling the broken ends apart, what kind of a result do you think you would get? You would get an enormous callus. Again, if you had a skin wound of the forearm and you kept pulling it apart every few days, what kind of a result would you get? It would finally heal, but with the formation of a large mass of scar tissue. That is the mechanism of the ankylosis. If you keep the parts perfectly quiet during the entire period of repair until the fragments unite, and then, and not until then, slowly commence motion, you will get a movable joint. On that plan of treatment repair takes place more normally, and without the excessive production of callus.

In the management of fractures of the olecranon, what do we do? Under the old splint treatment one of the chief dangers of fractures of the olecranon was in the dressing. With our plan, if you dress the arm out straight you get no trouble; if you dress it flexed at the elbow, you get no trouble. When you have a fracture of the olecranon all you need to do is just to make a nick through the skin and the triceps tendon and drive an eight- or ten-penny nail through the olecranon into the shaft of the ulna, and you secure perfect immobilization from the first day onward, without additional trauma to the bone. That is our method of extra-articular bloodless nailing of fractures near joints. That

is the easiest way to manage fractures of the olecranon. You can do the same thing with fractures of the condyles of the humerus or femur or of the malleoli.

The skiagram in this case shows that there is bony union between the ulna and the humerus. There is no bony union between the radius and the capitellum of the humerus. Pronation and supination are normal. If that whole joint were ankylosed, I should be in a better condition to get a satisfactory result than I am now.

After the usual preparation of the field of operation an Esmarch constrictor is applied high up on the arm by one of my assistants *before* he puts on his gloves. Formerly I made a vertical incision on the posterior surface of the arm, directly over the olecranon. I have since abandoned that procedure because of the fact that the contraction of the scar later often interfered somewhat with free motion in the joint. I now take two lateral incisions, one on either side of the olecranon—although at times only one such incision may be necessary, depending on whether or not it is possible to work freely through an external incision without injuring the ulnar nerve.

If there is any danger of inflicting injury on this nerve, the second or internal lateral incisions should be made. I always insist on finding the nerve, freeing it from adhesions, if any are present, and retracting it out of the field of operation, before proceeding with the further steps of the operation.

The length of this lateral incision will depend on how much of an area one wishes to expose. As a rule, an incision about three inches in length is sufficient, although there is no reason why it should not be made longer if necessary. The incisions are made about one-half inch away from the olecranon on either side, and extend through the skin and superficial fascia. The edges of the wound are then retracted widely so as to give easy access to the joint.

The interposing flap in these cases is taken from the aponeurosis of the supinator longus and from the fascia and fat on the inner side of the joint. The bases of these flaps are directed distally. The flaps are made sufficiently wide so as to cover the

freshened surfaces of bone, and long enough so as to reach across from one side of the joint to the other.

The ankylosis in the case of the elbow usually takes place between the olecranon process of the ulna and the humerus. This connection is carefully divided by means of a curved chisel, driven in laterally until free mobility of the joint is secured. In our earlier work we thought it advisable to separate the olecranon with its muscle attachments from the rest of the ulna and retract it upward so as to expose the interior of the joint. We no longer find this procedure necessary, except in unusual cases.

Sometimes there is bony ankylosis between the head of the radius and the lesser sigmoid cavity. When this union exists, it also is chiseled free, all exostoses are cut away, and perfect freedom of motion is secured. If necessary, the olecranon fossa may be deepened, so that the arm may be completely extended. Ample bone should be removed from the humerus and radius to permit of free flexion and extension *without force*, and the anterior capsule of the joint fully and completely divided.

Having secured the desired mobility of the joint and having freshened all bony surfaces, the next step in the operation is to place and secure the interposing flaps. These flaps are drawn into the joint and secured on all sides with fine chromicized cat-gut sutures. The ulnar nerve, which was exposed at the commencement of the operation, so as to protect it from injury, is now replaced in its groove and surrounded by fat. The remainder of the operation consists of the steps already described, such as closing the wound and applying the bismuth subiodid powder and dressings.

The elbow is immobilized at a right angle, and a posterior and lateral plaster-of-Paris cast embracing three-fifths of the circumference of the arm is applied. At the end of from three to six weeks the cast is taken off twice a day and passive motion is instituted. At the end of ten days, however, the cast may be removed permanently and the arm carried in a sling. [While a plaster-of-Paris cast is the dressing which we have used in this work most frequently in the past, at the present time we are

coming more and more to use the wire-gauze splint in its place. The latter is cheaper, and more neatly and quickly applied; its shape can at any time be altered and the splint can be used again and again on different patients and on different parts.] The patient is encouraged to make active and passive motion, and, if necessary, is ordered to carry sand-bags or other weights for the purpose of securing as much extension of the arm as possible. Massage may now be of service in increasing the function of the joint. Care must be taken not to fracture the olecranon. The passive motion should never be made severely painful or too prolonged.

In the case of the patient before us today we are told that he had a fracture of his olecranon, followed by bony ankylosis between the olecranon and the humerus. This statement of his is confirmed by the skiagram, which shows also that the coronoid process and the olecranon are much enlarged as a result of bone deposits upon them.

In order to expose this patient's elbow-joint we shall make the usual longitudinal incisions, one external and one internal, on either side of the olecranon, and about two inches apart. Through the internal incision we shall free the ulnar nerve and retract it out of the field, so that it will not be injured during the subsequent course of the operation. The next step in the procedure is the division by the chisel of the ankylosis between the olecranon and the humerus. We shall have to be very careful here not to produce any more serious injury to the soft parts than that which has already occurred, and, therefore, with great care, we drive the narrow curved chisel in between the olecranon and humerus in the line of the former joint, which is still plainly visible. We pass the chisel first through the internal and then through the external incision until, with very slight upward pressure on the forearm, the ankylosis gives way. We shall have to remove some of the excessive bone from this olecranon in order to insure normal extension of the arm. The head of the radius and its capsule need not be disturbed because pronation and supination are normal, which means the entire absence of adhesions between the head of the radius and the lesser sigmoid cavity.

We believe that we now have restored the anatomy of this joint to normal to such a degree that the patient will subsequently have a useful arm. All that remains to be done is to prevent recurrence of the ankylosis, and this end we accomplish by the interposition of one or two pedicled flaps of fat and fascia.

I have emphasized repeatedly in this clinic the necessity of working out the technic of these operations first on the cadaver before attempting it on human beings. The technic certainly must be thoroughly mastered in all its details before it can be practised successfully on living patients. The mechanics of these joint operations are fully as important as perfect asepsis. I always feel as though I do not emphasize that point strongly enough.

Now as to the preparation of the interposing flaps. I believe that for this patient we shall need only one flap, because there is an ankylosis only between the olecranon and the humerus. Therefore a single flap of sufficient size to reach clear across this point will be enough. We have removed sufficient bone from the internal condyle and from the olecranon to make room for the interposing flap, which we shall remove from the external (lateral) surface of the arm. This flap consists of the fascia from the upper portion of the supinator longus and from the lower portion of the biceps muscle. It will measure about three inches in length by one and one-half inches in width, and its pedicle will remain attached below, that is, the upper or proximal end of the flap will be free. This tongue-shaped flap is grasped with the forceps and is drawn backward and inward so as to cover all the raw bony surface on the posterior aspect of the humerus. The flap will lie between the humerus and the olecranon. It is drawn carefully inward and its edges are sutured with chromicized catgut to the connective tissue over the posterior edge of the internal condyle, where the ulnar nerve formerly lay. It is also tacked to the anterior edge of the base of the internal condyle and to the posterior surface of the external condyle, thus securing its permanent fixation in that position. That step practically completes the operation, and you will observe that every portion of the raw bone surface is now covered

by this flap and that there is no tension on it. Therefore, the ankylosis cannot recur and the flap must live.

We are now ready for the closure of the wounds. The deeper layers are approximated with plain catgut sutures, the ulnar nerve being displaced forward out of the zone of scar formation so that later it will not be compressed or constricted. The skin edges are approximated with horsehair. We never resort to drainage in these cases. A good-sized alcohol and phenol gauze dressing is applied, and the arm is put up with the elbow flexed at a right angle and the hand midway between pronation and supination. We shall not put on a plaster cast, but build, instead, about the arm a wire-gauze cage which extends from above the shoulder-joint to the finger-tips, and does not hazard the circulation in the arm in the least, a point which I insist always on mentioning because close attention to the preservation of a good circulation is the only way you can prevent the occurrence of an ischemic myositis and a subsequent contraction of the forearm tendons—the so-called Volkmann's contracture.

The arm will be kept perfectly quiet in that position for about four weeks, and then the patient will be allowed to use it. At no earlier time will forced motion be resorted to. We like this wire netting for making these splints, because with it we can make the splint of any shape, any slant, and any size at a moment's notice; and when finished, you can see through the splint and know that the support is just where it should be.

This operation is a slow one. It must be done with great deliberation and mechanical accuracy, because if the mechanics are correct and the technic is precise, a good result is a foregone conclusion; but if your mechanics and technic are faulty, then you can predict with certainty a failure. I have watched closely the literature on arthroplasty by various means for many years now, and always have found that our results with the pedicled fat and fascia flap compare favorably with those of other clinicians.

We have always used the *pedicled* flap of fat and fascia to interpose between all such adjacent bony surfaces, except in two cases, both arthroplasties on the knee. One of these two cases was an absolute failure, but not because of the operation or our

technic. When the patient, a hysteric girl of sixteen, left the hospital she had good motion in the joint to the extent of 45 degrees; but after she left our care, she simply refused to move the knee or to allow any one else to move it. The result was a recurrence of the fibrous ankylosis to the same degree as when she first came to us.

In the second case in which we used a free transplant instead of a pedicled fat and fascia flap there was considerable suppuration; but in spite of that mishap, the flap lived and the patient got a movable joint. He was a very thin individual. He had lost one leg as the result of a gasoline burn, and his remaining leg was badly crippled and flexed to a right angle. There was a bony ankylosis between all the bones of the knee-joint. He was a very unfavorable subject for arthroplasty; but we yielded to his insistence and operated, after having explained to him that we did not expect to be able to do anything for him except to straighten his leg. He wanted a straight leg, even though it was stiff, because then he could walk, whereas bent up as he was he could get around only in a wheel-chair. There was no tissue around the joint which we could use for an interposing pedicled flap of fat and fascia. Therefore, we were obliged to transplant a portion of fascia lata from his trochanteric region, with the result just stated.

In the case of the girl mentioned above, a previous operation on the knee had resulted in the formation of so much cicatricial tissue that the only flap which could be secured was from the fascia lata in the trochanteric region. On the whole, however, we find the pedicled flap has given us satisfactory results; therefore, why should we change to any other method, such, for instance, as the artificial interposition of foreign material or transplanted flaps? We are convinced that when surgeons appreciate fully what an arthroplasty means, they all will adopt the live-flap procedure and that then their results will be equally as good as ours.

[*Note.*—The result in this case was splendid. The wounds healed by primary union. When the patient left the hospital about five weeks after the operation he had free flexion and extension within an arc of about 45 degrees.—ED.]

NAILING OF FRACTURE OF SURGICAL NECK OF HUMERUS AFTER AN UNSUCCESSFUL ATTEMPT TO SECURE UNION BY BONE TRANSPLANTATION

HISTORY

THIS patient, a girl of eleven years, entered the hospital December 24, 1913, because of inability to use her right arm. On October 17, 1913, the patient fell from a horse, striking the ground with her right shoulder. She immediately had pain in the shoulder, which was greatly accentuated on attempts at motion. Half an hour after the accident the arm became quite swollen, and twenty-four hours later ecchymosis was visible. She held the arm suspended and flexed at a right angle at the elbow. This relieved the pain. A sling was not used because it gave her pain. She had no chills or fever. Two days after the fall a doctor was consulted, who said that no fracture was present, gave her a liniment to apply, and bandaged the arm.

This treatment was followed for one month. Then the patient noticed that her right arm was shorter than her left, and that she could not abduct it. She could not raise her hand to her head, chin, or left shoulder, nor could she draw the right arm backward. A skiagram was then taken, and it showed a fracture of the humerus with the lower fragment luxated upward and in front of the upper fragment.

About November 20, 1913, the fracture was reduced by an open operation and the arm dressed with the elbow at a right angle in a cast. Four days later another skiagram was taken, and it showed a slipping outward and upward of the lower fragment of the humerus. One week later the cast was taken off, the stitches removed, and the cast reapplied. December 4, 1913, two weeks after the first operation, a second operation was

performed and a short transplant from the crest of her tibia was inserted between and into both fragments. The arm was again dressed in a cast. About a week later another skiagram was taken, and it showed that the transplant had slipped out of the upper fragment into which it had been inserted. She now comes to the hospital, brought by her parents, who ask for relief of the deformity and a restoration of the lost function in her arm.

COMMENTS AND OPERATION

DR. MURPHY (January 3, 1914): We do not need the history in this case. The skiagram is sufficient. It shows the fracture of the surgical neck of the humerus and also discloses the fact that an effort has been made to apply a bone transplant to immobilize the fragments. The transplant is embedded in the lower fragment, and its upper end, which was originally embedded in the head of the humerus, has escaped from the humerus and is projecting outward into the soft parts. The doctor who did the transplantation is a good man, and perfectly frank in acknowledging his error in using a bone transplant for this purpose. He wrote us a letter something like this:

"Dear Doctor: I attended the Congress and saw you do some of these bone transplantations; then I went home and tried to do likewise." I must say, in respect to him, that he did the operation very well; although he performed two open operations on the shoulder and took a transplant from the tibia, it all healed without infection, which shows how thorough his asepsis was; but he selected the wrong case. A recent fracture is not a case for bone transplanting. That procedure should be resorted to only when there is a failure of union. All that was necessary in this case was to reduce the fracture, bring the bone around in position, and put nothing in it. At present, as the result of what he actually did do, we have a softened humerus head from the bone absorption which has taken place since the former operation, nearly four weeks ago.

We do not know just what we shall have to do. Probably we shall put in a nail. We are not going to take out the transplant until we know that we have something else, for instance



Fig. 176.—Fracture of neck of humerus. Bone graft adherent to distal fragment only. Position of the fragments before operation.



Fig. 177.—Grafted bone sawed off flush with end of shaft of humerus. Head fastened in position to shaft by two wire nails. Callus formation well advanced.

a nail, in there to support the head of the humerus and fasten it to the shaft.

The purpose of a bone transplant is to aid and stimulate regeneration of bone. Therefore it should be used in cases of non-union of a fracture, and not in recent fractures where conditions usually are favorable for union, particularly in a bone fractured through its cancellated portion. Our present practice in the management of fractures of the surgical neck of the humerus has been followed in about ten cases. We nail the fragments together after first securing a good apposition by an open operation. In nearly all these ten cases on which we operated the head was luxated out of the glenoid cavity. In this patient today it is not luxated. The shaft, however, is displaced upward and the upper end of the transplant has forced its way through the deltoid muscle and can be palpated just beneath the skin over the deltoid. We shall need to be very careful here not to traumatize the deltoid, because if we do traumatize it, our patient is likely to lose the power of abduction in her arm. The management of the deltoid is the really serious part of the operation in this case. The transplant, you see, lies quite close to the skin.

We shall make the incision along the anterior border of the deltoid, commencing at a point just to the outer side of the acromion and extending downward on the arm, a distance of about three inches. There is the long head of the biceps in view. There is the acromion. The humeral head has not been thrown out of the glenoid cavity, although it is slightly rotated. The shaft of the humerus lies to the outer side of the head. Flexing the arm and rotating it outward, the upper end of the shaft is thrown into view. There is the transplant! it is firmly embedded in the shaft—so firmly that it cannot be pulled out, which shows thorough work on the part of the surgeon who made it. Therefore, we shall cut it off flush with the shaft. Note that the periosteum on the transplant is soft and necrotic and yet there is active osteogenesis in the transplant, as shown by its firm fixation to the shaft. We are going to have some difficulty in getting the lower fragment around to the proper angle to the

upper because of the contraction of the surrounding muscles which has taken place and must be overcome. Two and a half months have elapsed since the accident. I must stretch these muscles slowly, and I do it by means of the bone skid, using the latter as a lever to elongate these muscles by forcing the broken ends into contact with each other. What we need are time and patience, because these fragments must be placed in apposition and without tension, too, so that the apposition may be maintained naturally. Now, you see, at last the fragments go together easily and stay in position.

The muscle which has suffered most from this stretching is the deltoid. The deltoid does not stand much abuse. When a man falls against or strikes his shoulder on a stone wall or any other hard surface, the accident often results in a paralysis of the deltoid lasting for sixteen or eighteen months; so you can imagine what may happen to the functional capacity of this muscle when it is traumatized as this one has been.

We shall secure the fragments in this position by driving a wire nail through the shaft from its outer aspect and up into the head of the humerus. Now, you can see that the humerus moves as one piece. The wound is closed in the usual manner, just with horsehair skin sutures. The arm is dressed in extreme abduction—to keep the head firmly in the glenoid cavity and thoroughly relax the deltoid—and is held in that position by means of a wire gauze splint extending clear across the patient's back and down the arm to the finger-tips. The forearm is placed in right-angle flexion with the arm.

Let the record show that the graft was firmly fixed to the neighboring structures; that its periosteum was dead and had slipped off the graft, notwithstanding the fact that the graft had become integral with the shaft. Have new blood-vessels and osteoblasts penetrated into it by means of the Haversian canals and led to reorganization of the graft and fixation to the shaft by new bone formation? No, because the first operation was done in December, just about a month ago, which is too short a time for such an organic union to be established.

I want to compliment the doctor for the careful work he did

in putting in the graft. The wound stayed sterile, and the graft healed in nice shape; but the proper indication for grafting was not present. If the humerus had broken two inches lower down, a large graft would have been desirable and would have been a splendid success. The doctor underestimated his skill in his letter to me.

Let the record further show that we exposed the fragments easily and that with the aid of the skid the fragments were swung around in position and a nail inserted; that the projecting portion of the transplant was removed; and that the arterial pulsation in the wrist is perfect, thus showing that the axillary artery was not injured by the manipulations during reduction.

[Healing of the wound was by primary union. The patient made a most excellent recovery in every respect. She left the hospital on February 9, 1914, about five weeks after the operation, and there was good movement in the shoulder-joint.—Ed.]

FRACTURE-DISLOCATION (SUBCORACOID) OF HEAD OF HUMERUS. REPOSITION OF HUMERUS HEAD INTO GLENOID CAVITY AS AN AUTOPLASTIC GRAFT WITHOUT VASCULAR ATTACHMENTS

HISTORY

THE patient, a man of fifty-two, entered the hospital January 21, 1914. His present trouble dates back to January 9, 1914, twelve days ago, when an automobile in which he was riding turned turtle and threw him out of the car. He received several body bruises. The right knee was tender to pressure. The perineum was lacerated. There was an injury in the right shoulder region. His right arm hung down limply. It pained him so much that he did not try to move it. His open wounds were dressed, and two hours after the accident an anesthetic was given him; the right arm was manipulated in an effort at reduction, splints were placed on the inside and outside of the arm, and a temporary dressing applied. On January 19, 1914, an x-ray picture was taken which showed a fracture and dislocation (patient thinks) of the right humerus near the shoulder-joint.

At present the shoulder is swollen, painful on motion of the arm, and ecchymosis extends down the internal surface of the arm, apparently following the biceps. There are also ecchymoses on the adjacent chest-wall. There is a luxation of the head of the humerus into the subglenoid fossa.

COMMENTS AND OPERATION

DR. MURPHY: What date was the accident?

INTERN: January 9, 1914.

DR. MURPHY: The x-ray tells the story, as you see. This man has had a fracture of the neck of the humerus and a luxation

of the humerus head into the subglenoid space, a comminuted fracture of the upper end of the bone. The tuberosity attached to the supraspinatus is torn off of the shaft and lies as a separate fragment in front. Here you get a very good view. You see that the head lies way over in front. If you have never done this operation, you do not realize how much trouble is ahead. We have gone into such cases before and do not relish them. Our plan is this: We have made our incision internal to the long head of the biceps, internal to the coracoid process to which is attached the biceps, and external to the head. Then we shall free the capsule of the joint. We should like to go between these two muscles, the biceps and the coracobrachialis, in this position. But it will not give sufficient space. So we shall cut off the tip of the coracoid process, leaving the coracobrachialis, the short head of the biceps, and the pectoralis minor attached to it, and reflect them downward. That gives us excellent space. Then we shall expose the glenoid cavity and get it ready for the reception of the head. Now, then, it is still possible that we can lift the head of the humerus back into the glenoid cavity without detaching from it that part of the capsule which is still firmly attached to it. If we cannot do that, then we shall do as we have done in other cases, lift the head out on the table, freshen it, and put it back into the glenoid cavity. There it will unite with the shaft of the humerus. I say it will unite because it has done so before, and made a good, movable joint. That means using it as an autoplasic graft. In these cases the bone is dead and has to be reorganized. No matter whether it is dead for a minute or for a month, it is just as dead at the one time as at the other, and the living tissues of the whole bone must be regenerated. There is where the head should lie, down on the inner side of the glenoid cavity. We can take it out, freshen its broken end, and put it back; it unites by primary union. Then all is well. But of course there is danger of an infection. If you get an infection, such a transplanted head becomes a sequestrum. [Incision.]

There is, you see, still a good joint in here. This bone has been out of place for about fifteen days. I am going through the



Fig. 178.—Oblique fracture of the surgical neck of the humerus, with subglenoid dislocation of the head of the humerus. Comminution of the upper end of the lower fragment. Radiogram made before the operation.

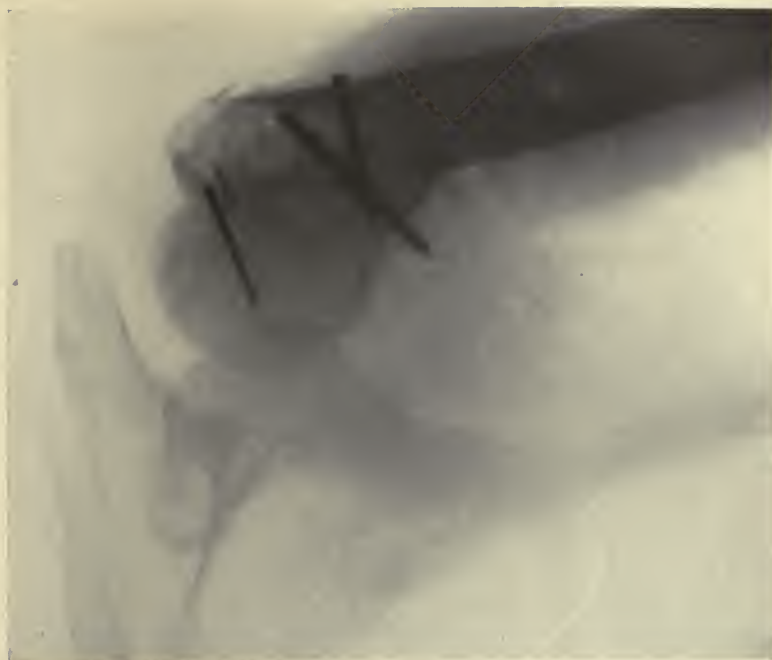


Fig. 179.—Oblique fracture of the surgical neck of the humerus, with subglenoid dislocation of the head of the humerus. Comminution of the upper end of the lower fragment. Radiogram made after open reduction of the subcoracoid dislocation of the head of the humerus. The plate shows the accurate approximation of the fragments and their retention in proper position by three wire nails.

deltoid now. See what a powerful man he is! See how thick that muscle is! Now we are cutting down on the fibers of his pectoralis major. There is the acromion process. Here is where we must displace these tissues outward. Here the fibers of the pectoralis major belly are attached. There is probably a hematoma just beneath here. It does not appear that the circumflex vessels were involved. There is a blood-filled pocket under the head. There is the upper end of the lower fragment. Now we come to the capsule of the joint. There is fluid, serous fluid, in the joint. Here is one of the upper fragments of the tuberosity which is attached to the supraspinatus. Here is the glenoid cavity. I am going to let you see it in a minute. See it there?

Now comes the really difficult part—getting out the upper fragment, which is lower down and to the inner side of the upper end of the shaft. I shall detach the pectoralis major a little lower. Remember that the vessels and nerves lie all to the inner side; that the circumflex vessels which supply the shoulder come down low and behind. There is the acromion. There is the head of the biceps. There is the upper fragment. There is the margin of the upper fragment. There is the humerus head over there. The vessels lie close at hand. I must cut the muscular attachments here of the latissimus. See! that powerful muscular attachment must be freed before I can bring out the head. Now I think I can luxate that head back into place with the skid. Now I have it back in place, you see. There is the lower fragment lying behind. See! we skid it back into position. There is the coracoid process. That I am going to bring back and reunite with a phosphor-bronze wire suture. There it is! Can you see it? An operation like this is a big job. It is not the kind of a job which is conducive to the operator's longevity. He gets the reward for his labor in seeing it effectively done, if not in any other way.

Let the record show that the fracture was found just as it was interpreted from the x-ray, namely, that there was, first, a fracture of the tuberosity, and, second, a fracture and an internal luxation of the head into the subcoracoid fossa; that the fracture was first exposed by an incision internal to the long head of the

biceps; then the glenoid cavity was exposed and no new tissue found present in it; that then the lower end of the upper fragment was exposed and freed from its attachment to the latissimus which was binding it downward and inward; that the head was then elevated by a process of rotation and by the assistance of the skid and was slipped over the margin of the glenoid into the cavity; that the other fragments were restored to proper position; that after the two fragments were brought into apposition two nails were used to hold them in position and a single nail was used to reattach the tuberosity; that the fragments were then accurately in position and the outer layer of the deltoid was sutured over them; that the fragments remain in perfect position without apparent tension.

You will see that there was no part of the operation which was really difficult except the nervous tension on one in doing it, without getting the hands into the wound. To one who is trained in abdominal surgery it is a little difficult to get along without using one's hands and without putting the hands in the wound. Abdominal surgery is practically all handwork. This bone surgery is all instrumental work. I believe that head has no vascular attachment remaining. So far as the ultimate outcome is concerned, if the wound remains sterile the amount of vascular attachment does not matter much. If the wound remains sterile, this patient will get a union almost or quite as good as though the circulation was preserved. That has been the case with those patients whose pictures I just showed you. The heads of the humeri were once put out on the table and were then put back. Now is the most favorable time to do the operation, namely, about the fifteenth day, when all the injured tissues are in the granulation stage of repair, and, therefore, relatively resistant to infection.

[The wound healed by complete primary union. The patient left the hospital February 23, 1914, about four weeks after the operation. He was at that time wearing his arm in a sling, and putting the shoulder through passive movements, but was not allowed to move it actively much. The x-ray, as is usual in these nailed cases, showed the fragments in excellent position, but without much callus as yet.—ED.]

COMPOUND FRACTURE OF LOWER THIRD OF FEMUR, LOWER END OF UPPER FRAGMENT PENETRATING KNEE-JOINT AND RESTING UNDER PATELLA; OPEN APPosition WITH LANE PLATE

HISTORY

THE patient, a railroad man, entered the hospital with the following history: On January 27, 1914, while riding on the brake step at the end of a box-car, another car collided with it, the draw bar was driven in, and the two cars, in coming together, caught the patient's right leg and arm between them. He was thrown to the ground, a distance of about eight feet. He then crawled away about sixteen feet from the track, and lay there until assistance came. He was carried to the depot and a doctor was called, who said he had a compound fracture of the femur. The bone protruded through the skin, but not through his clothing. Dry dressings were put on the wound, and the hemorrhage stopped. He was then taken to a hospital, where an attempt was made to reduce the fracture without an anesthetic. Splints were applied to the side of the leg and extension with a weight of 32 pounds was made and left on for two days. Then he came here.

Since coming to the hospital bichlorid dressings have been used daily. His arm shows ecchymoses from the shoulder to the elbow, but he has complete use of it.

COMMENTS AND OPERATION

DR. MURPHY (February 14, 1914): Here is an x-ray of his case. It shows what a lamentable condition this is. Here is the x-ray as he came in. Do you see that fracture? Do you see what has happened? There has been a fracture from above

downward and backward and a luxation of the upper end of the lower fragment backward, and the lower end of the upper fragment is luxated forward and downward and has penetrated the joint in the intercondyloid groove. It is also compounded on the inner side. (See x-ray.) This fracture occurred on what date?

INTERN: January 27, 1914.

DR. MURPHY: Today is the fourteenth of February—eighteen days have elapsed. We have waited until now for two reasons. Although he came in to the hospital about the tenth or eleventh day after the injury, we got him ready for operation at the time, notwithstanding there was an ulcer of the skin. There appeared to be no infection in the fracture, but there was an infection in the skin. When the skin was in a condition safe for operation he developed an acute pharyngitis, and that complication has postponed active procedures until now. Now we are in the position where we are hovering between two dangerous alternatives, one of letting this leg go longer as it is and become fixed in that malposition; and, second, of opening up those tissues in the presence of an infection of the skin, which means the possibility of carrying microorganisms into that wound. There is the further possibility that he has some of that purulent infection left in his throat, and there may be still in his blood some of the streptococci which were in his throat. But amid all these dangers we are trying to keep the middle road and steer between the perils on either side. If the present position of the fracture becomes permanent, you certainly will be in trouble with your patient. If you get an infection, your patient certainly will be in trouble; but we are trying to avoid both these unpleasant eventualities.

We are going to do an open reduction here. If we could once get the lower fragment in contact with the upper fragment, it would stay in pretty good position of itself, because the gastrocnemius muscle pulls that lower fragment sufficiently backward so that it will stay in position at the proper angle in that direction and give a result not half bad. If he gets an infection, he will probably have to have an amputation; he will then lose his leg and possibly his life. All these possibilities were duly



Fig. 180.—Compound fracture of lower end of femur. Marked overriding of fragments, the upper fragment lying in front of the lower and behind the patella after penetrating the knee-joint.



Fig. 181.—Compound fracture of the lower end of the femur. Fragments replaced in proper position and fastened with Lane plate. Muscles united with phosphor-bronze wire.



Fig. 182.—Same. Lateral view.

weighed and it was decided that the best course to follow was to wait until this time to effect the reduction.

There is the x-ray plate made this morning. You can see there has been, notwithstanding the severe traction, no reduction of the deformity; that the lower end of the upper fragment rests on the external condyle. That is fine. I had rather have it rest on the external condyle, because I wish to go in on the external side. He has an infected laceration on the internal side, and I wish to keep as far away as possible from that laceration.

We have here rather a nasty looking leg. There is an ulcer here on the inner side—an open ulcer. That is where the lower fragment was driven through the skin. In doing all this work of open reduction we endeavor to follow the directions given by Mr. Lane, which are what? To make all manipulations with instruments, not touching any of the patient's tissues with the hands or with the instruments at the points where the latter have been handled. That is the key to good results. You remember the case he did in this clinic? Remember the large incision and all the blood? He did nothing in wound toilet but close the skin. There was a hematoma formed; it discharged blood, but went right straight along and healed without a single mishap. In doing this line of work the reason so many operators get bad results is not that the method is wrong, but that the method is not properly carried out by those surgeons. *The work must be done with instruments* and not with the hands. It is not an easy matter, especially for one who has been accustomed in abdominal surgery to use the hands for everything, to get into the habit of using only instruments.

We have put a constrictor on this leg. Why? Because the bleeding is going to be profuse. We put it on to save time and also to save the vital resistance of the patient by conserving his blood. You can see that the difficulty and danger in this case lie in getting the upper end of the lower fragment freed of its vessels and placed in apposition with the upper fragment. There will be difficulty because, in the first place, there is shortening of the limb, due to contraction of these muscles during the last eighteen days. Therefore it will have to be elongated by

traction and then the lower fragment made to pass into position. Once it is in position, if we can get it there, then the manner of insertion of the plate will be important. The probabilities are that we shall use a six- or eight-screw Lane plate. Possibly we may use a Milne plate and possibly we may use our own plate. The Milne plate gives an angular reënforcement which I may try to take advantage of.

[Incision.]

There is fluid, you see, where my incision has opened up the joint. There is the lower end of the upper fragment. There you see the vastus externus.

Let the record show that there is cloudy, purulent exudate in the knee-joint and on the end of the upper bone fragment, notwithstanding his low temperature.

Let the record further show that there is some pathologic material here in the wound, evidently purulent; that this material was sent to the pathologist for examination.

Here is another piece of bone taken out with some of the same purulent material for pathologic examination. See the large quantity of fluid which is flowing out! It was pent up between the two fragments. I will see whether I can locate the upper end of the lower fragment.

Let the record show that there is a considerable quantity of fibrous tissue anterior to the lower fragment, and separating it from the upper fragment, and that this tissue is evidently a portion of the joint-capsule.

Here is the mass of tissue in between the two fragments. I am dividing it at its lower side. It looks like a continuation of the joint-capsule. Careful inspection does not show vessels in it, nor a nerve. I am using only the upper end of the bone skid. I will see if I can split this lower attachment. We have not yet completed the reduction, but we have succeeded in getting the bone fairly well in line. Now we have an excellent apposition. I want you to see the perfection of that reduction. You can scarcely see now where the fracture occurred. Let me put this screw in and then I will show it to you. I want to rotate that plate just a little bit. I want it to fit the reduction exactly. I

am putting the plate just on the side of the joint. I fix the plate to the bone with the forceps. Now I insert the screws. The bleeding is not excessive, although there is some. There will be a great hernia of the vastus externus muscle there if I do not do something with this aponeurosis, so I will just bring the aponeurosis edges together with phosphor-bronze wire of the finest size. You must never drain where you put in a foreign body, unless you make up your mind that you are going to take out the foreign body. This will be a continuous suture in the aponeurosis. You lessen thus the hernia and you lessen the opportunity for hematoma formation. The bleeding now has practically all stopped. There is the vastus internus as it comes over in front of the field. See! I am well anterior. That aponeurosis came together nicely. We have succeeded in getting through our operation without entering the old wound. I am keeping my fingers away from the part of the suture which is to touch the tissue.

Let the record show that we first came upon the lower end of the upper fragment and found that it had penetrated the capsule of the joint; that we then elevated the bone so as to expose the upper end of the lower fragment; that when the latter was exposed we used the skid to swing it around into position.

You can readily understand from the double curve on that skid what happens when the end of the lever goes up. It pulls on all these structures and does not let your fragment go until you have the reduction completed. There are only three special instruments used in all our bone work, and this is one of them

Let the record further show that we did not expose the infected cutaneous field at all in our procedure; that it was well protected with collodion before the operation and remained so protected during the reduction and the entire operation; that the reduction was not difficult; that great care was exercised not to disturb the vessels in the popliteal space, and we believe that we have completely avoided them; that an eight-screw plate was inserted; that the wound was closed with silkworm gut and covered with collodion.

In addition to the dressings that leg is put in a cast. You

may think that is unnecessary. No, it is very necessary. It is necessary because, on account of the proximity of the fracture to the knee-joint, the gastrocnemius, in pulling down on the lower fragment, may spring the screws. Now we put that in extreme abduction and keep it there until it has united. I shall cut a hole in the side of the cast for dressing the wound.

We succeeded in bringing that limb into splendid position and, I think, with a minimum of manipulation, possibly not quite as artistically as Mr. Lane would do it, but still we put it in good position and got the plate inserted nicely. Unless we have an infection from the suspicious material which we found in there, we should get primary union. The plate, if properly put in, is the best instrument that ever was invented to make permanent the reduction of fractures.

It is the little things usually which make the difference between success and failure. It takes very little to break that plate, for instance, and then our work would have been in vain. Look at the secure grasp the doctor has on that leg; that holds the plate safe. We first get the cast spread out and then slip it under the leg, so that it comes clear up to the hip and embraces the body to the costal arch and down to the toes, so that no tension is put on the screws at any time. Although the screws are supposed to hold the fragments in apposition under ordinary leverage, it is easy to put too much tension on them—just as easy as it is unnecessary.

The results which we have been having with the Lane plate have been fairly good—I do not say perfect, because we have had some cases that were not successful. We have had some infections with the plate, something we rarely have in any other clean field and should not have. They are becoming fewer and fewer. If the plate can be put in in a perfectly aseptic way, it makes the ideal method of holding bone fragments in apposition. It requires conscience as well as technic not only in asepsis, but also in the use of the instruments. You see I had to determine the position of these bone fragments in order to make sure I was making my incision right. In place of feeling the fragments with my hands, I gained the same information by pressing

on the skin with the back of the razor blade. When I could not find the distal fragment the temptation was great to put my finger in the wound and locate the upper end of the lower fragment. Yet it is easy enough, as you could see, to acquire the proper technic with training.

The effect of the plate on osseous union is still an open question. It seems to me that we are getting more non-unions than we used to have, and I think they are due to two things,—two essential factors,—namely, that in fractures of the middle of the shaft or of the middle three-fifths of the shaft of long bones, if the fragments are too completely immobilized, we are likely to have a failure of union; and that in fractures into or near joints we immobilize too little, get too much motion, and, consequently, too much callus and sequential limitation of motion. We have incontrovertible examples of perfect approximation with the open operation and with the screw plate in which there has not been the slightest effort at union. Such instances undoubtedly are becoming more frequent. I am speaking now chiefly from my own experience; but my own experience is not an average experience, because those cases that heal up uneventfully I do not get, as a rule. They do not have to come back for a bone transplant. I am speaking from a local experience and I am sure we are having more non-unions than we had thirty years ago. It is, I think, because we get too perfect immobilization of these long bones.

Remember that the Germans, when they put on a cast for a long bone fracture, usually put the patient in ambulation. That much activity keeps up a certain irritation at the ends of the bone which I think stimulates union, so that they have fewer non-unions than we have at present. I am speaking now, of course, only of fractures in the middle three-fifths of the shafts of long bones. The only exception to this rule is the fracture of the lower fifth of the humerus. You get a larger percentage of non-unions with fractures in the lower fifth of the humerus than in any other end-bone location in the body. Why? Because there is no cancellated tissue there to speak of. The lower fifth of the humerus consists of compact tissue right out to the end of the

condyles. Remember, too, that the neck of each condyle is made up of compact bony tissue, just like the compact bony tissue of the shaft of the humerus. Non-union does not occur so readily where you have a fracture away from the end of the humerus, because you have a large quantity of cancellated tissue there, and fractures in cancellated tissue rarely result in non-union. It is in the fractures through the more compact bony tissue that we fail to get union.

In the treatment of fractures in the neighborhood of joints the possibility of a non-union does not figure so largely in our calculation. One produces, when the fracture is near a joint, the greatest possible immobilization of the fragments and the joint. Why? Because mobilization means callus and a large callus in the neighborhood of a joint means a limitation of motion. Remember, I am speaking now only of fractures when they lie near a joint. Any additional trauma to a joint which has been injured always involves great danger of ankylosis. Formerly, I remember, it was the practice in fractures about the elbow-joint to take them out of the cast from the fifth day on and start passive motion. Now it is our practice to keep them in the cast until the end of the fifth week and then start passive motion. Why? Let us look back at the conditions present in fractures at joints five, six, seven, or eight days after the fracture. Suppose you start motion, you tear all these healing tissues apart. What do you get? More connective tissue and more bleeding; the more bleeding, the more callus; the more callus, the more limitation of motion. If you had an incised wound of the skin of the arm and at the end of the fifth day you took out the stitches and started to pull the wound apart, what would you get? A great big scar. And you fail to apply this common knowledge of visible repair to the repair of hidden tissues like bone.

Formerly we used to apply some form of irritation to the ends of the fractured bone, but that did no real good. You must put some material which is osteogenetic in between the two fragments. The easiest method is the autograft, a piece of bone taken from the man's own tibia and put in position across the line of separa-

tion. Then you get a union provided you keep the fragments absolutely immobilized.

Now you see that in one class of cases I suggest you should get more motion between the fragments, whereas in the other class you must not have motion. Similarly if you make a transplant and then immediately start active or passive motion, what forms around the transplant? Connective tissue. The transplant then slips out from the other portion of the bone and consequently you have a case of non-union. You must keep that limb immobilized until the blood-vessels from the living bone have penetrated into the Haversian vessels of the transplant and carried with them the osteoblasts and osteoclasts which are necessary for the reconstruction of bone. If you have a fracture and make a transplant and then get your patient up in five or six weeks, you will have a failure of the graft to unite.

We had one case of non-union of a graft at its upper end, because it lay too near the knee. Why? Whether we did not keep on the cast long enough or whether we were unable to immobilize the knee enough, I do not know. The upper fragment slipped away about one-third of an inch from where it was put in contact with the transplant and the result was that we failed to get union. We had no absorption of the bone, but a non-union. We shall finally put in a bone graft and immobilize it properly, and I think we shall get a splendid result. Here are the pictures of the case. (See CLINICS, 1912.) Here, you see, is the sarcoma specimen which was removed. We opened into the joint, took out the bone entirely from the inner side, and put in a new piece of bone. I want to show you the non-union. Look! See where the fragment separated from the bone. You see there the end of the fragment, and there the pseudoarthrosis. It has all healed in, and I think it is nearly two and one-half years since we took out the sarcoma. It was a year and a half after their doctor had told them that an amputation of the leg was all that could be done to save her life that we saw her here. The people would not consent to an amputation, and possibly they were right. But if the proposition of transplantation had been made to them originally when the tumor was only a small

growth, and when the growth was encapsulated, the chances of final success would have been greater. That is where we are going to benefit our patients in the future. Whether or not we ultimately lose this particular patient does not matter, so far as the future of this work is concerned, if we make the diagnosis early. The people will not consent to an amputation, but they will consent to having a piece of bone taken from one tibia and inserted in the other. They would rather die than have an amputation; so this operation is a benefit so far as sarcoma is concerned, particularly in the slowly growing type of chondro- or fibrosarcoma, and possibly the medullary type, as shown in so many of these tumors.

[The operative wound healed by primary union and he has complete firm bony union with almost perfect bone conformation (see *x-ray*). The wound caused by the compounding is still discharging slightly (May 8, 1914). He is getting a little passive motion, carefully carried out, but has not yet been permitted to try to walk on the limb.]

CARCINOMA OF RIGHT HIP, METASTATIC FROM THE BREAST. EXCISION. BONE TRANSPLANTATION TO FILL THE DEFECT

HISTORY

THE patient, an unmarried woman, aged thirty-four, entered the hospital December 19, 1913, because of inability to use the right leg in walking. About six and one-half years ago, in 1907, the patient noticed in her right breast a hard, small, movable lump, about the size of a marble and not painful. It grew very slowly and without pain. In October, 1911, four years later, she commenced for the first time to have pains in the breast shooting to the right shoulder and down the right arm. November 25, 1911, her doctor applied a paste to the lump in the breast. The paste remained on for three weeks, at the end of which time the tumor was taken out without cutting. She had continuous, excruciating pains during these three weeks. The wound healed with no more pain or trouble. On June 26, 1913, two years later, the right breast was removed by a surgeon without apparently any reason for so doing so far as the patient could see (?).

In October, 1912, she suddenly developed pain of a severe stinging character on the internal surface of her right thigh, one and one-half inches below the groin. The pain continually increased in severity, and did not migrate or radiate. The limb became weaker, and she says she stumbled easily. Wet or damp weather accentuated the pain. The pain was also increased by walking, causing the patient to walk with a limp. Rest in bed did not make the pain disappear. Heat relieved it somewhat. She noticed that pressure increased the pain.

June 4, 1913, while stepping from an elevator, suddenly she

slipped, caught herself, and then sat down; but the slight fall was not sufficient, she thought, to cause a fracture. She was carried to a doctor's office and later was taken to a hospital. For three weeks hot applications were made to the limb, which was swollen, stiff, and very painful, and she says it could not be flexed at the knee. The symptoms grew less, although the pain continued. On July 2, 1913, an *x*-ray plate was made which showed an intracapsular fracture of the femur. On July 11 the limb was put in a box-cast with extension and counterextension. This apparatus was kept on for six weeks. On September 15 she went home and the doctor said the fracture had united. The knee was less painful and more flexible, but she has not been able to walk since the accident, because the right leg gives way under her.

Six years ago she had a mild attack of typhoid fever. Twelve years ago she had a "nervous breakdown." Twelve years ago she also had a fall on her back. She suffered with severe backache following the accident, but was not confined to bed. She also has had continuous headaches, referred to "the base of the brain," for the last five years. The backache was relieved by treatment by an osteopath, who said that one vertebra was out of place.

COMMENTS AND OPERATION

DR. MURPHY (December 20, 1913): The breast tumor was first noticed six and one-half years ago, the patient being then twenty-seven and one-half years old. According to her story to me, she slipped and fell, in June, 1913, and struck her knee so severely on the floor that she also injured her hip. She had the tumor removed from her breast, first by paste, two years ago. Six months ago the whole breast was removed by operation, she being then thirty-four years old. If there was a carcinoma in her hip, she could get a pathologic fracture of the bone while walking on a perfectly smooth floor. If, on the other hand, she slipped and struck her knee severely, then she could have fractured a normal femur. The severity of the trauma, however, was clearly not sufficient to fracture a normal femur in a girl of that age. Furthermore, pain had been present in it for eight months

preceding the trauma. It is, therefore, a pathologic fracture of the neck of the femur from a metastatic carcinoma of the neck of the femur. This diagnosis is verified by the skiagram, which shows the absorption of the bony matter of the trochanteric and cervical zones of the femur as the result of the cancerous infiltration.

[Incision.]

You can observe that the neoplasm involves a considerable portion of the femur. She would not consent to an amputation, preferring to die rather than to lose the limb. If I can take out all this mass and then transplant in its place a piece of bone from her tibia, I shall do so.

If this neoplasm is a metastatic carcinoma, it lies here in the usual location. If it is a metastatic sarcoma, this is also the usual location. If this growth is a carcinoma, the patient's life is more endangered by it than if it be a sarcoma.

As you can see, there is an enormous enlargement of the femur. There is a failure of union in the fracture below. I have made a U-shaped incision here, and split the tissues, so I could pick up one muscle after another and turn them aside until I come to the neoplasm. If it is a cancer, I shall make an attempt to remove it and replace it with an auto-transplant.

We are now down to the fascia lata behind. I am separating the fascia lata from the trochanter. There is the gluteus maximus. I wish to preserve the strength of this fascia lata as much as possible, because the support will be needed. There is the gluteus medius muscle. We shall cut it loose from the greater trochanter and turn it forward and upward. Here is a part of the neoplasm, apparently. It is a compact, hard mass. All of that upper end of the bone is soft. I shall take out the tumor by a wide dissection, so I must enlarge the opening to the side and cut all these muscles. The tumor is still fairly well encapsulated and has not yet invaded any of the neighboring structures; but we must get out every possible vestige of it with its capsule. Now we are down on the shaft to normal bone, and are ready to commence work on this capsule and to open this joint to see what lies inside. We now have all these structures nicely pre-

pared and exposed. On looking in now you can see the neck and the head of the femur. We shall open the capsule by a circular incision. Here is the little piece of bone which is all that is left of the head and neck. Here is the neoplasm invading the bone. On examination with the knife the blade goes through the infiltrated bone like putty. We shall commence this excision from above and proceed downward. Now we are coming to the trochanter minor, and you can see where it is broken here. There is the obturator internus and piriformis which come around here inside of the trochanter. Finally, here we have out the encapsulated tumor and we shall send it at once to the pathologist so we can get an early microscopic report as to whether it is a sarcoma or a carcinoma.

[Demonstrates the specimen to the clinic.]

There is the pathologic fracture in the specimen. From the gross appearance I cannot say positively whether it is a carcinoma or a sarcoma. I wanted to take out all this dubious tissue with the affected bone. There is a perfectly normal medulla, you see, where we sawed through the femur below the tumor. Shall we do the graft now? No; there are too many chances of infection. I shall close this great cavity now and put in the graft at a second operation. If that were my leg, I should do just what this patient did—refuse amputation.

Now we shall go back again to our field of operation. We shall first clean out that joint-cavity. That is a very pretty pathologic fracture. Evidently the neoplasm also invaded the head, it is so soft and friable. I shall, therefore, take it out. There is the capsule. We are elevating these muscles again so as to get things in fairly good shape for the subsequent transplantation. We are closing this capsule over the joint, so I shall have a relatively clean joint-cavity to come back to if I ever do the transplantation.

The pathologist reports that the tumor looks like a metastatic carcinoma. That diagnosis will fit in with the assumption that the breast neoplasm which she first noticed when twenty-seven and one-half years of age was a carcinoma. She made the statement that there was carcinoma in the family. I take it that the

doctor who made the microscopic examination of the tumor excised two years ago found the growth to be cancerous, but did not tell the patient so. It is not uncommon to conceal from a patient, who comes of a cancerous family, the true nature of her growth, because in this manner the patient's hope is kept up.

There is the gluteus medius muscle which I am refixing here to the neighboring muscles in its normal relationship to the central zone formerly occupied by the bone, where it will become attached to the bone in case I do a subsequent implantation of bone into the hip.

Let the record show that the tumor appeared entirely encapsulated and was removed *in toto* with its capsule, together with most of the capsule of the joint; that the head was found to be involved in the disease and was removed; that the ligamentum teres was also infiltrated by the neoplasm, but that there was apparently no neoplasm in the acetabulum; that the joint cartilage appeared normal in every way, and that the cancellated tissue of the diaphysis of the femur showed nothing resembling either sarcoma or carcinoma.

[Dr. Murphy then exhibited the specimens, pictures, and roentgenograms of several cases of sarcoma of the hip operated upon by him.]

DR. MURPHY (January 28, 1914): I have had this patient prepared to make a transplant from her tibia and I shall reopen the scar tissue which has replaced the cavity from which the bone was removed in her right hip. Here, you see, is a white, fibrous, suspicious-looking mass, and it may be a return of the carcinoma. Here is also a thickened and somewhat infiltrated medulla in the end of the shaft. They both look suspicious to me. Dr. Bevan, what do you think about them?

DR. BEVAN: They look like malignant tissue.

DR. MURPHY: Very well; we shall not do the transplantation then till the pathologist has examined them. Therefore, I shall close the wound temporarily with deep silkworm-gut sutures. If the pathologist's report is satisfactory, that is, if he reports no carcinoma present in the tissue, we shall make the transplant on Saturday, January 31.

TRANSPLANTATION OF UPPER THIRD OF FEMUR (SAME PATIENT)

COMMENTS AND OPERATION

DR. MURPHY (January 31, 1914): This case we had in the clinic on January 28, 1914, and removed a specimen from the remaining diaphyseal marrow of the femur which the pathologist reported showed merely cancellated tissue without the slightest evidence of malignancy; so we are going to transplant this morning a piece of this patient's tibia to replace the head, neck, and trochanter of her femur.

[Incision.]

There is the end of the bone which we exposed at our previous operation in December. Now we cut through the remnants of the capsule and commence to see the acetabulum. There is fluid present now in the acetabulum close to where the head lay. The fluid appears purulent. She has had no temperature, however. The question is, whether it is not just a sterile, serosanguineous accumulation of fluid such as one finds in the peritoneum occasionally. I am dilating the capsule which was sewed over the acetabular cavity at the previous operation. That is where I have to insert the upper end of the graft. I am dividing the capsule at its lower angle, so I can slip it over the graft later. Here is where I have to implant the graft, in the medullary cavity of the femoral diaphysis. Let me see how long a piece of bone I really shall need—seven inches.

We now have the cavity prepared to receive the transplant. Remember, I excised the head and the trochanter originally because of a secondary carcinoma in them. Dr. Bevan asked me last Wednesday if this was an experiment and I said, "No." It is not an experiment; but we know that a carcinoma in any place in the body is likely to recur, therefore it can recur here. We also know that if we transplant a strip of bone here from the tibia the graft will live and grow into the hip articulation and give carrying support to the trunk and a movable joint.



Fig. 183.—Carcinoma of the femur (before operation). Carcinoma of head, neck, and metaphysis of femur, secondary to carcinoma of the breast. Pathologic fracture of neck of femur. Diaphysis of femur shows no pathologic changes.



Fig. 184.—Carcinoma of the femur (after the first operation). Head, neck, and metaphysis of femur excised.



Fig. 185.—Carcinoma of the femur (after the second operation). Bone graft from the tibia replacing the excised upper end of the femur. Note lower end of the graft planted in the medullary cavity of the femur and the upper end resting at the edge of the acetabulum. Following the taking of this radiogram the leg was dressed in still greater abduction to bring the upper end of the graft nearer the center of the acetabulum.

Third, we know that in carcinoma of the breast you may have only a single metastasis present in the body, and, after removing it together with the original tumor, may never have any recurrence. We have taken out osteosarcoma in this location for the same indication. Sarcomata are more frequently seen in the upper end of the femur than carcinomata, according to our observation. This patient was wise in not consenting to an amputation—wise, because the mortality from hip amputation is much greater than from the excision of the trochanter and head. I did not do primary transplantation at the time of the first operation, because of the extent of the field of operation and the danger of sequential infection. I waited until the area was all healed and full of granulation tissue, because granulation tissue is enormously more difficult to infect than is normal healthy tissue.

Let the record show that when we cut down on the end of the femoral diaphysis we found there a large fibrous mass, not organically connected with the bone, but capping the end of it and derived from the tissues outside of the bone and easily separable from the latter.

[Incision made over the tibia.]

In removing the transplant from the crest of the tibia we never make the incision directly over the prominent portion of the tibia. We are taking a much thicker and longer graft here than the average. I have to measure to see how much of this tibial crest must be taken off—seven inches. I have taken a longer piece than I shall keep. Now I am getting ready to slip the graft into the acetabulum.

The report on the smear from the little pocket of fluid found in the acetabulum is negative for bacteria, so it was just the non-bacterial leukocytic pus such as we find in peritonitis of the defensive type that we used to think was so dangerous. The tissue we took out with the fluid is reported as granulation tissue without any bacteria present.

There will be a Buck's extension placed on this limb, so as to take the pressure off the graft, this being exactly the way we treated the hip sarcoma cases, the pictures of which will be

thrown on the screen. The extension will be maintained to keep the graft from pressing too hard against the cartilage in the joint during the process of repair, and from being thrown out of plumb in consequence. In a little while all this transplant will become adherent both to the end of the femur and the soft parts and then the graft will develop further in the acetabulum.

That the patient does not get a return of the carcinoma *in loco* is our chief concern, this being the first case of the kind we have had. Of course no one can say yet whether or not she will get a return of the carcinoma; but her chances for not getting a return are just as good as with an amputation. By doing nothing at all in such cases the mortality is 100 per cent. Following operation there is at least some chance for recovery. We have in the hospital a woman who has a metastatic carcinoma in the trochanter of her femur, which was first noticed about four and one-half years after she had her breast tumor removed by amputation. She has at the present time not the slightest evidence of metastasis anywhere else in her body. That means what?—that if that trochanter had been excised in the beginning when the disease was first noticed, encapsulated as such metastases are in the beginning, she would have had just as good a chance for a cure as she had before the involvement of the femur. This woman has now had the metastasis to her knowledge for something like one and one-half years. It is six years since she had the carcinoma in the breast removed.

Everybody who is familiar with the metastasis of cancer knows that many of the cancer-cells which leave the primary site are taken up by phagocytes and finally overcome. If you can give nature just a little assistance, you can *hope* for a cure. That is one reason why we use sodium cacodylate in such cases—because we believe it stimulates the phagocytic cells. It is not supposed to have any direct specific effect on the cancer-cells themselves. The *x*-ray is also of value in cases of cancer, producing a hyperplasia of the connective tissue which in turn causes a strangulation of the cancer-cells. After treatment by massive doses of *x*-ray such as we can now give easily and accurately with the admirable Coolidge tube the operation biopsy will show

the breast with a center of cancer and a hard coffer-damming of connective tissue all around it. You probably do not cure the cancer by this means, but you render it relatively harmless.

This excision operation should make a material difference in the cancer mortality, because patients will consent to excision when they will not consent to amputation. If we could attack these bone metastases early, as we do malignant disease in other parts of the body, then we could get as good results as we get, for instance, in operations for cancer of the breast. The patient may live a long time after this operation without any manifestation of disease and you have avoided an amputation. The only objectionable feature to the excision and subsequent transplantation is that the tension on the operator is too great. You have to watch everything which is going on—not only your own manœuvres but also those of your assistants, because the slightest non-sterile contact infects the graft.

A twenty-pound extension will be kept on. It is five weeks now since we excised the hip. It is three days since we took out the specimen of granulation tissue and sent it to the laboratory, fearing that it was malignant. I did not want to try to implant a graft if cancer tissue was still present at the site of operation. The microscope showed nothing but connective tissue, therefore we are justified in putting in the graft in a conscientious and surgical way, taking every precaution that the patient may not be harmed. We did not make the graft at the first operation because of the extensiveness of the original operative procedures. I had to detach several of the muscular attachments at that time and then reunite them with sutures. They united so beautifully that it was difficult for me just now to find where the original bone lay. I also had to be on the watch for the sciatic nerve in one place in my dissection.

Let the record show that we first exposed the cut end of the femur, which was covered by a mass of connective tissue, firm, hard, and almost cartilaginous; that this tissue was about one-half inch thick, and covered the end of the bone very much like a cap, going out from the periosteum, evidently from where we had peeled the periosteum off around the end of the femur; that

this cap was not attached to the bone, but lay quite free; that the medulla of the bone was excavated and reamed out in the regular way to receive the graft.

Let the record further show that the acetabulum was opened and found to contain about an ounce of a fluid resembling pus, which was thin and watery, with no inflammatory reaction in the tissues around it; that the capsule of the hip-joint was opened but was not resutured; that a slit was made in it for the admission of the upper end of the graft; that cultures and smears were made from this suspected fluid and that the smears showed no microorganisms present, only leukocytes and tissue cells; that a graft six and one-half inches long, after it was finally trimmed, was taken from the crest of the opposite tibia and inserted into the reamed-out medulla of the shaft of the femur; that traction was then made on the leg by rope and pulley and, with our skid used as a lever, the transplanted bone was slipped into the acetabulum.

[The microscopic section of the excised upper end of the femur showed the tumor to be a *cyndric-cell carcinoma* diffusely infiltrating the cancellous tissue. The wound healed by primary union except for the formation of a hematoma, which had to be aspirated (350 c.c.). Up to the present time (May 8, 1914) there has been no return *in loco* and none elsewhere in the body. The patient is in splendid condition, and is able to walk around the hospital with the aid of crutches.—ED.]

CARCINOMA OF MALE BREAST

HISTORY

THE patient, a man aged fifty, entered the hospital February 18, 1914. About October 1, 1913, he was struck near the nipple of the right breast by a bottle which was thrown at him from a distance of about ten feet. The breast immediately became slightly swollen, painful, and red. He had no chills. By the end of a week the pain, swelling, and redness had disappeared. The breast returned apparently to normal. October 25, 1913, while visiting a patient, he leaned against a bed-post, the right breast coming in contact with it, and shortly afterward the breast became painful. Ever since then he has pain whenever anything comes in contact with the breast. Since October 25, 1913, a small lump began to develop to the outer side of the nipple, at the site of the original injury to the breast. This lump has gradually increased in size, until it is now about as large as a pigeon's egg. It is firm, rounded, regular in outline, not fixed to the chest-wall or to the skin. There is no retraction of the nipple; no pitting of the overlying skin; no enlargement of the glands of the axilla.

COMMENTS AND OPERATION

DR. MURPHY (February 21, 1914): We shall first excise this tumor and examine it before we decide on the final operative procedure. Two elliptic incisions, going wide of the mass, are made, and the tumor and underlying tissues down to the pectoral muscle aponeurosis are excised. The tumor is fairly firm. Bisecting it, we note that the gross appearance is that of a carcinoma. Carcinoma of the male breast is a much more serious proposition than carcinoma of the female breast, because there is not the relatively clean-cut encapsulation in the former which you see so commonly in the latter. The reason for this fact is

that the connective tissue of the male breast does not offer the same inflammatory resistance that the connective tissue of the female breast does. Therefore, the percentage mortality is much greater in carcinoma of the male breast than it is in carcinoma of the female breast. Fortunately, carcinoma of the male breast is a rather infrequent occurrence. We shall send this specimen to the pathologist for an immediate report before proceeding further.

While we are waiting for the report, let us go over the history and see how it corresponds with the usual course of development of carcinoma. About four or five months ago the patient was struck with a bottle. The traumatized area became red, swollen, and painful, but there was nothing to indicate that it was a serious condition in the breast. These symptoms subsided in two or three days and disappeared entirely within a week. Three weeks afterward the breast again became sensitive and he noticed this small swelling. Now, then, if this had been a hematoma in the breast, the result of a trauma, the swelling would have remained for three or four days, but the soreness would have continued for weeks. Here the soreness all subsided with the swelling, but both returned after three weeks, and finally he noted the presence of a definite tumor mass. That is the clinical course of carcinoma, the sequence of trauma to the breast. The trauma in the breast, however, is always of a peculiar type. The etiology of carcinoma in almost every other place in the body, except in this location, is repeated, recurrent irritations. In the tibia, on the lip, in the stomach, in the liver—carcinoma is the sequence of repeated irritations. *In the breast, however, it is very frequently the sequence of a mild trauma of single occurrence.* Note the difference! That is the only place in the body where carcinoma takes on that character. Sarcoma, when the result of a trauma, is usually the sequence of a comparatively severe trauma of single occurrence. For instance, a man is playing ball and the ball slips through his hand and strikes the humerus; or he is playing golf and the golf-ball strikes him on the shin; or he strikes his leg against a chair or a table. The trauma is never severe enough in these cases to produce a

fracture. *If a trauma does produce a fracture of a bone, it never causes a sarcoma at the seat of the fracture.* We have never seen a sarcoma occur from a fracture, but we have seen a large number of pathologic fractures result from sarcoma.

We recently had in this hospital two very striking cases of pathologic fracture as the result of metastatic carcinoma in the femur, and another from a primary sarcoma in the femur. The first patient was a woman who had an amputation of the breast five years preceding the pathologic fracture. Some time after the breast excision she developed pain in her hip, which was called "sciatica," as is commonly done in these cases. To get relief from the pain she began to use crutches. One day she caught her foot on a threshold, and, to save herself from falling, she seized the handle of the door. She did not even fall; and yet it was found that she had a broken femur in the region of the trochanter as a result of this slight trauma. That is not the history of a fracture of a normal femur. The other patient, a younger woman, had an amputation of the breast for carcinoma. About a year later, getting out of the elevator, she stumbled and fell to her knees and was unable to rise. When she was examined by the doctor it was found that she had a broken femur. She had had pain, however, for a number of weeks previously in the hip and thigh, and she was told she had sciatica or neuritis, when the fact was that she had a metastatic carcinoma in the trochanteric zone of the femur, which was easily demonstrated by the x-ray. The trauma was merely severe enough to fracture a pathologic femur, but not severe enough to fracture a normal femur.

The case of sarcoma of the femur occurred in a young man, a railroad employe, who, while running between cars, slipped and nearly fell. In order to save himself he grasped a hand-rail on the car, and, as he told us, "strained his leg." It kept hurting, but he continued to work for some time. Finally, he had to quit work on account of the severe pain in his thigh, which had been diagnosed as "rheumatism." He did not work from February to July. On July 3 he resumed his work, but only for a few days, on account of the pain in his leg. This pain

became more and more severe and he finally went to bed and was treated for "sciatica." Finally, turning over in bed one day, he fractured his femur. A normal femur will not break under such conditions. That was evidence at once of pathologic fracture, and yet the condition was not diagnosed. An operation was later performed for the treatment of the fracture, because it would not unite. Of course it did not unite. The skiagram, when made finally, showed clearly what it was—a sarcoma. A sarcomatous or carcinomatous fracture never unites.

There is practically nothing to do for these patients after you have made the diagnosis; but if you analyze the history closely and the clinical course of the disease, you can always make the diagnosis, very frequently even without the skiagram. You make the skiagram as a confirmatory procedure, just as you use the laboratory; but it is from the clinical course that you make the diagnosis.

If the tumor in this case today proves to be a carcinoma, we shall perform the radical operation, removing the entire breast and cleaning out the axilla. If it proves not to be a carcinoma microscopically, then we shall accept the pathologist's opinion, but we shall put a large black interrogation point after it, because we have learned that the clinical course of the disease plus the gross pathologic findings in many of the cases is just as valuable in differentiating between malignancy and benignancy as are the microscopic findings. When the clinical manifestations and the gross pathologic appearance leave you in doubt, then you usually have the report coming back from the laboratory with a question mark, showing that the pathologist, too, cannot interpret the case with certainty. In other words, when the diffusely infiltrating and metastasis-forming growth characteristic of malignancy shows clean-cut in the gross appearances, you can make the diagnosis often just as well from that as with the microscope.

Where the nature of the neoplasm is obscure, either because of the excessive resistance of the patient or the exceptional character of the neoplasm, then we may be unable to make the diagnosis from the gross appearances. One variety of tumor

which often leads to a difference of opinion between the clinician and the pathologist is the purulent cyst, which is malignant on its inner side; that is, has a carcinoma developing from the epithelial cells lining the cyst-wall. The pathologist often sends back a report that it is not malignant, when, on the basis of your experience, you feel that it is malignant. The majority of these cases get recurrences after you remove the cysts, so that finally you reach the conclusion that they are very apt to be malignant, the pathologist's report to the contrary notwithstanding. We had one case in which we operated four times. At the first operation the microscopist thought the case was not malignant. However, the growth recurred and recurred, and finally, at the fourth examination, the pathologist decided it was malignant. We carried the patient along for something like four or four and one-half years before the fatal outcome. There was no doubt in my mind at any time that it was a malignant case, that it was a carcinoma which originated in the inner lining of the cyst-wall. I merely mention these cases to accentuate the fact that the microscopic examination of tumors is merely a support for the evidence you get from the clinical course and gross examination, and that the clinical result is the final court of appeal as to whether the lesion is malignant or benign. When you operate very early in these malignant cases then you do not have the clinical results, recurrences, and metastases as the final court of appeal, because you eradicate by the operation all the malignant tissue. In such cases it is difficult to refute the microscopic diagnosis, either of benignancy or malignancy. But when the tumor recurs, the subsequent clinical course proves beyond doubt that we had not an innocent cyst, but a malignant one.

The pathologist just reported that this is a cylindric-cell carcinoma; therefore, we shall do the radical operation.

[The operation for removal of the breast for carcinoma was described in the February CLINICS, p. 179.—ED.]

As an additional safeguard against recurrence, this man will have x-ray treatments, beginning about the fifth day from now. These treatments are given with an aluminum plate interposed between the ray and the skin, because that protects the skin from

burning by filtering out the rays of slight penetration. One rarely has a serious irritation of the skin unless the patient is extremely feeble. If you have not an aluminum plate, sole leather can be substituted. It is the "soft" rays of slight penetrating power and, therefore, slight therapeutic value which cause burns. The aluminum plate allows the more penetrating "medium" or "hard" therapeutic rays to pass through, while it cuts off the dangerous "soft" rays.

PATHOLOGIST'S REPORT (Dr. Zeit): The microscopic examination of the tumor from the breast shows it to be a scirrhus, cylindric-cell carcinoma, apparently originating from the milk-ducts. The lymph-nodes removed show no trace of metastasis, either grossly or microscopically.

[*Note.*—The wound healed by complete primary union. Eight days after the operation he developed an acute tonsillitis and a metastatic lymphadenitis in the left cervical glands, which required incision and drainage. The healing of the wound was in no wise interfered with by this unexpected incident, nor by the patient's chronic morphinism—20 grains a day. He left the hospital March 9, 1914, with a good and useful arm.—Ed.]

OSTEOMA OF THE HEAD OF THE FIBULA; REMOVAL OF TUMOR AND BONE; TRANSPLANTATION

HISTORY

THE patient, a girl, aged fourteen years, entered the hospital January 6, 1914, with the following history:

About fifteen months ago, when taking off her stocking, the patient felt a lump half the size of an English walnut situated two inches below and on the outer side of the right popliteal space. It has never caused her any pain. It is harder to palpate at present than formerly, owing to the presence of an increased amount of tissue (*fat?*) over the tumor.

In January, 1913, she was in bed for two days with tonsillitis. In August, 1913, she noticed that the right leg in the region of the tumor appeared to be larger, but she paid no attention to it until six weeks ago, when she measured the leg and found it to be one inch larger in circumference than the left leg at a level two inches below the patella. She does not remember having injured the leg. It is not tender to touch and has never been inflamed. The tumor is hard and apparently fixed to the bone.

COMMENTS AND OPERATION

DR. MURPHY (January 7, 1914): This history is not of any special significance other than that it tells us the mode of onset of this trouble, thereby eliminating infection as a possible etiologic factor. When I first palpated this tumor, I took it to be a neoplasm of the tibia, because it lay directly behind the tibia; but when I saw the skiagram, I recognized at once that it was not connected with the tibia in any way, but that it was a tumor of the head of the fibula, involving all the head of that bone and extending inward behind the tibia for three-fourths of the

width of the tibia and so close to it that no space between the tumor and the bone could be distinguished by palpation. [See x-ray pictures for lateral and anteroposterior views both before and after operation.—ED.]

Our diagnosis is an osteoma. The tumor is well encapsulated. It does not infiltrate the surrounding tissues at all. Its consistence appears to be fairly uniform in the skiagram, so that it is not a case of osteitis fibrosa cystica. Just notice how well defined the tumor wall is. Why? Because it consists of compact bone. There is no breaking down in the center of this tumor, no cavity. There is no indication of malignancy.

This tumor originated in the upper end of the fibula, and it has grown toward the popliteal space. The external popliteal nerve has not been involved in any way, because she has perfect function of the foot. Remember this nerve winds around the head of fibula from behind forward, and it is fortunate that this tumor grew inward instead of outward, because it therefore spared the foot function. The contents of the popliteal space have not been injured in any way; and while the posterior tibial artery is out of harm's way, the anterior tibial is going to be hazarded materially in the operation. Indeed, it may be necessary to ligate this artery. We have gone over this case very carefully before deciding on the course of procedure we are going to adopt this morning. We have concluded, because of the benign nature of this tumor, to resect the upper end of the fibula and transplant a portion of bone from the crest of the opposite tibia, thus making a new upper end for the fibula.

Remember, however, that while these tumors have every appearance of being benign, there is always the possibility of a malignant change having taken place somewhere in the tumor mass, a fact which can be determined only by making a microscopic examination of all the tumor. However, if it be a malignant lesion, we feel assured that removing the tumor at this time will have the desired result, possibly avoiding a fatal outcome and preserving a useful leg. If this were simply a bone cyst or an osteitis fibrosa cystica, we should break down the canopy of the tumor, curet out all the soft granulosomatous tissue from its



Fig. 186.—Cartilaginous exostosis of head of fibula growing backward into popliteal space and lying behind tibia.



Fig. 187.—Cartilaginous exostosis of head of fibula.



Fig. 188.—Same as Fig. 189. Lateral view.



Fig. 189.—Excision of head of fibula, with cartilaginous exostosis. Autotransplant of bone from crest of opposite tibia to replace the defect. Anteroposterior view.



Fig. 190.—Cartilaginous exostosis of head of tibia similar to the pathologic condition in the fibula described in the text. Lateral view. This tumor was removed without transplantation.



Fig. 191.—Cartilaginous exostosis of head of tibia. Anteroposterior view.



Fig. 192.—Same patient as Fig. 193. Anteroposterior view, showing the exostosis removed.



Fig. 193.—Cartilaginous exostosis of head of tibia excised. Lateral view.

inside, being particular to remove the soft tissue lining, and fill the cavity with bone removed from the crest of the patient's tibia. In other words, we adopt the same procedure that the dentist employs in filling a tooth. He cleans out the cavity and then fills it. In that instance the cavity is not obliterated by new tooth tissue, but in these cyst cases the bone transplanted into the cyst acts as a transplant, and if there is osteogenesis, the cavity is eventually obliterated by the formation of new bone. We have had cases of bone cysts involving the lower end of the radius, the phalanges, the tibia, the femur, and the humerus. Some of these cases we treated as outlined above, and in the others we resected the diseased portion of the bone and did a transplantation, the line of procedure adopted depending entirely on the extent of the disease and the particular part of the bone involved, and the factor of weight-bearing. Whenever possible, we clean out the cyst cavity or cavities and fill them with bone. In one of these cases, a simple cyst of the lower end of the radius, we utilized a portion of the cyst-wall. One of our earliest cases of bone transplant was an osteitis fibrosa cystica of the upper end of the humerus and the result was a splendid one. (See October, 1913, CLINICS.)

The case today will be the third case of tumor of the bone in which we have removed the tumor and transplanted bone. One of these cases was a chondrosarcoma of the upper end of the femur. (See December, 1913, CLINICS.) The other was a metastatic carcinoma in the upper end of the femur. (See this issue, p. 557.) In another case, a fibromyxochondroma, the upper end of the femur was involved. It was removed, but the transplanting has not yet been done, the wound not having entirely healed. There is still some discharge, although not purulent in nature.

Our patient is now ready for operation. A constrictor has been placed high up on the thigh, because we must have a clean field for this work. We make the incision to the outer side of and behind the fibula, just in front of the outer head of the gastrocnemius muscle. Retracting the wound margins, you see we get a good exposure of all the field of operation, not only of

the tumor, but also of the external popliteal nerve and the vessels. By a blunt dissection we bring the external popliteal nerve clearly into view and we shall retract it backward, keeping it out of harm's way all the time. The tumor involves about two inches of the upper end of the fibula, and it is encapsulated. We want to remove this tumor without involving the knee-joint in any way. By using the periosteotome to free the soft tissues, we can bring the bone and the tumor clearly into view. Now we shall pass the chain saw underneath the fibula, just below the tumor, and divide it. Now we can swing out the tumor very easily by just turning it forward and inward over the anterior portion of the tibia. Nothing has been traumatized—none of the neighboring structures has been traumatized or torn. All these tissues are left in good shape and there is no reason why we should not have a good result in this case. We shall ream out the fibula for a distance of about an inch, getting it ready for the insertion of the transplant, which we shall remove from the crest of the opposite tibia. We never put a constrictor on the leg from which we remove the transplant, and we make the incision not over the crest of the tibia, but to the outer side, over the tibialis anticus muscle. We split the aponeurosis of this muscle and then retract the muscle downward and outward so as to expose the crest of the tibia. Then we split the periosteum on both sides of the crest with the scalpel, marking out the length of the transplant we desire to remove. With the motor saw we are going to remove a transplant about three and one-half inches long, by one-fourth inch by three-eighths inch, with its periosteum. As this piece of bone is freed from above it is grasped with two sequesterum forceps, so that it cannot be lost. The incision in the muscle aponeurosis is then closed with catgut and the skin with horsehair.

We are now ready to place the transplant into the fibula. We shall drive the lower end down into the enlarged medullary canal of the fibula, and the upper end of the transplant is placed into the small pocket made for it on the external surface of the tibia, just underneath the tuberosity, thus maintaining the normal anatomic relationship of the fibula to the tibia. We ex-

pected to have to nail the transplant to the tibia to maintain its fixation in this position, but we find that it is held so securely in this pocket that the nailing need not be done.

That completes the operation. Before closing the wound we shall test the external popliteal nerve with the faradic current, and on doing so we find that the muscles supplied by it respond, showing that the nerve has not been divided or injured. The deep structures will be approximated with plain catgut and the skin with horsehair. The usual dressing will be applied to the wound and the leg placed in a wire-gauze cage. In about two weeks, if all goes well, that is, if the patient does not develop any symptoms of an infection or discomfort in the leg, the first dressing will be done. The patient will be kept quiet as long as possible to allow the regeneration of the upper end of the fibula to take place. After five or six weeks a leather leg cast will be applied, and the patient allowed to be up and around on crutches, bearing little weight on this leg, however.

[*Note.*—The microscopic examination of the tumor showed it to consist of bone surmounted by a cartilage "cap," a so-called cartilaginous exostosis or osteochondroma.

The wound healed by primary union. The patient left the hospital February 7, 1914, one month after the operation, and has not yet returned for reëxamination. We expect to get a practically perfect functional result in this case.—ED.]

PENETRATING ULCER ON THE LESSER CURVATURE OF THE STOMACH; RECURRENT HEMATEMESIS. CHRONIC PERICHOLECYSTITIS. POSTERIOR GASTRO-ENTEROSTOMY. OCCLUSION OF PYLORUS BY USE OF THE LIGAMENTUM TERES

HISTORY

THIS patient, an unmarried woman aged thirty-two, entered the hospital December 10, 1913, with the following history: During the fall of 1896 the patient was treated for anemia, but did not remain in bed. In the spring of 1897, when she was seventeen years old, she complained at times of a sharp pain in the epigastric region one hour after eating, the pain lasting until the next meal. This pain gradually increased in severity for a period of two weeks, at the end of which time she had the distress almost immediately after eating. Liquid or solid food, whether hot or cold, made no difference with the pain. During this period the patient one day vomited bright red blood twice within fifteen minutes, approximately two quarts in amount. The patient thinks this was in the month of April, 1897. She went to bed with an ice-bag to the epigastrium, remaining in bed for about fifteen days. While in bed she received as diet, on the doctor's order, only a tablespoon of milk four times a day for the first ten days, thereafter a glass of milk three times a day, and salines per rectum. For the following year she was on special diet, did very little work, and apparently recovered entirely, so far as she knew.

In 1901 the patient complained occasionally of indigestion and of pain in the epigastric region one hour after eating. This pain was not always constant. In the spring of 1902 the patient was again treated for anemia and palpitation of the heart. She also

had gas formation and pain immediately after eating. She lost in weight, had severe frontal headaches, constipation, and nervousness. After six months, with the aid of a tonic, these symptoms gradually cleared up. In 1908 she had typhoid fever, was in bed for five weeks, and recovered without any complications. Since the attack of typhoid fever she has been very constipated and has been compelled to take a laxative every day; she has also had severe headaches. In 1909 she noticed a raw, sore, burning feeling on the left side, over the iliac fossa. At times a hard lump could be felt which was very sore on pressure. The soreness does not radiate. The lump, however, would disappear after taking a large dose of laxative, after which gas would be noticed in the intestines and flatus would be passed.

Since 1911 she has complained of a dull, lead-like feeling of pressure in the epigastric region about one hour after eating, with gas in both stomach and intestines. This gas causes pressure against the heart, she thinks, and consequent palpitation. Immediately after eating she belches gas and food is regurgitated. In March, 1913, while the patient was acting as surgical nurse in the operating-room, she suddenly became pale, dizzy, and nauseated, the room went dark before her eyes, and she all but fell. At 11 P.M. of same day she felt as if blood were filling the stomach; some stomach-contents which she regurgitated tasted of blood. For several days afterward she noticed dark blood in the stools. She did not vomit during these days. From this time the patient has complained of palpitation and nervousness upon the slightest exertion. One hour in bed is all that is necessary for relief during such attacks.

Since March, 1913, the patient has had a dull feeling of fulness in the stomach. There is much gas in the stomach between meals and eating seems to help get rid of it, perhaps because she belches most after eating. She has often resorted to the use of the stomach-pump for relief. On November 1, 1913, she had a dull pain in the epigastrium, was dizzy, and vomited some dark blood. There was blood in the stools for the next three days and the patient felt faint. She remained in bed for



Fig. 194.—Ulcer of the duodenum, with practically negative x-ray findings. The stomach shows no trace of the bismuth meal administered five hours previous to the taking of the radiogram, showing that there is no delay in the emptying time of the stomach, as is often the case in duodenal ulcer. The head of this bismuth column may be seen in the lower ileum, in the right lower quadrant of the picture. The cecum, transverse colon, sigmoid, and rectum still contain portions of the bismuth meal administered twenty-nine hours previously.



Fig. 195.—Ulcer of the duodenum with practically negative *x*-ray findings. The stomach is distended with a Rieder bismuth meal administered immediately before the taking of the radiogram. It shows a large stomach, slightly dextroposed, but otherwise negative. The colon contains a similar bismuth meal given twenty-four hours before the taking of the radiogram.

three days on a liquid diet, taking a bismuth and acacia preparation three times a day.

At the present time the stomach feels somewhat less distended than formerly, but there is a full feeling after eating even a liquid diet.

Past history: She had measles at seven, no complications. Had mumps at twenty-two.

Menstrual history: Began at fourteen, irregular, from five to six days' duration, scanty. Has had dysmenorrhea during the first day since 1908. Very often she has a sharp pain in the pelvic region lasting from five to six hours about one week after menstruation. There are soreness and a bearing-down sensation a few days preceding menstruation. Three or four years ago, while bending over suddenly, the patient felt a sharp pain somewhere in the pelvic organs on the left side. The soreness lasted for two weeks. The stomach trouble seems to be accentuated at the time of menstruation.

COMMENTS AND OPERATION

The history is almost more than classic. It includes, you see, symptoms of hemorrhage, and, furthermore, symptoms of the collapse which follows severe hemorrhage. You will note she had three attacks of typical hemorrhage in which she fainted. There can be no question that this woman has an ulcer. The only question is, what kind of an ulcer? Has she a small, round, peptic ulcer which we shall be unable to find at operation, or has she a large, round ulcer which will be easily palpable through the gastric wall? Is the ulcer in the stomach or is it in the duodenum? Now, doctor, I wish you would read again the main points in connection with the disturbances which follow her meals. Do they come immediately or later?

INTERN: About an hour after eating.

DR. MURPHY: See! The distress comes within the stomach time-zone rather than within the duodenal. The duodenal period is a little later—from two to two and one-half hours. [Left paramedian incision.] We will just enlarge that incision a little toward the lower angle to give us a larger and better field.

Here is the pyloric region coming readily into the field. Here is an adhesion between the pylorus and the side of the gall-bladder. Here is the gall-bladder—look!—bound into this mass instead of lying free. Now we shall examine the stomach and see whether the stomach shows evidence of a large indurated ulcer or whether it shows evidence of the presence of a more superficial peptic ulcer. Here is the ulcer-bearing zone of the stomach where you have 84 per cent. of these lesions occurring (Mayo). Here is the lesser curvature, which I can mobilize clear up to the cardia. I will bring it all into view. Here is the dome of the stomach in which there rarely is an ulcer. Now we must palpate and see whether we can feel an ulcer anywhere. There, you see, is the pyloric vein. There is the pylorus lying just outside it. Here is a very much enlarged lymphatic gland. You can see it slip through my fingers. Now, then, we shall follow the duodenum down. It is fixed. The gall-bladder is firmly bound to it. Now I am palpating the pancreatic side of the duodenum.

Let the record show that the head of the pancreas is slightly enlarged; that it is not white; that all the glands behind the duodenum are enlarged. Let the record further show that there is organic union by scar tissue between the gall-bladder and duodenum.

I think I can show this all to you. There—look!—is the organic union between the gall-bladder and duodenum. See the gall-bladder! It is the white variety, the whiteness being due to scar tissue, thus showing that there has been an infectious process here. That is very pretty. This adhesion we must free. Now I am separating the gall-bladder from the duodenum, so that I can follow down and palpate the entire gall-bladder which I have now in my fingers.

Let the record show that the gall-bladder is adherent to pylorus and duodenum, compressible, and free from stone.

There is no evidence of a lesion in the duodenum. Let us see about the stomach. As we come back each time we run across that set of glands again. Those glands would not be involved by an ulcer farther back. Very beautiful! There are

the glands: see them there in my fingers! See them slide under my fingers! She must have some lesion in this zone, because these glands have no other reason for being enlarged. Here is the duodenum, distal to the pylorus. There is no evidence here of ulcer. Let us pass to the inner side. Here is the inner side of the lesser curvature. Now let us go to the upper curvature. I think I can demonstrate it to you before I am ready to close the wound. There it is—see it! Here is a small ulcer not larger than a dime and movable. The lesser curvature is not thickened, but is perfectly normal. Now I come back to this ulcer. There I can see the tip of the induration. I came near missing it entirely on account of its small size. That is the kind which bleeds. It is the kind from which my preceptor, Dr. O'Reilly, died. It is not on the pylorus. I should say it is about an inch above the pylorus. Now the question comes up at once—is it near enough to the pylorus to interfere materially with the outlet of food? Will you risk a future stenosis or will you do a gastro-enterostomy? These are the questions which come up in one's mind immediately. My idea is that she has had ulcer recurrences for years. If we leave this stomach alone, she will continue to have recurrences; therefore she should have a posterior gastro-enterostomy and, at the same time, a closure of the pylorus of the stomach by wrapping it with the ligamentum teres. We shall constrict the pylorus with the ligament after we have done the gastro-enterostomy.

There may be two lesions here. The glands and adhesions are evidence of a duodenal lesion not gross enough to be palpable; and we have shown a gastric lesion to be indisputably present. Bringing this loop of small gut up in this manner, putting tension on the ligament of Treitz, you bring into the field the first portion of the jejunum. We shall bring up this loop and get it ready for approximation. I put it up in this position and there let it stay. We shall expose the posterior wall of the stomach under this vascular arch in the greater omentum. Dividing the lesser peritoneum, as I do here in this non-vascular zone, we are able to spread the cut edges as much as we need. There is a line of adhesions toward the pylorus. Here is the lowest point

at which I can make the approximation. We shall divide the gastric serosa a little lower down. We shall displace into the greater peritoneal cavity this portion of the stomach, which lay originally in the lesser peritoneal cavity, and shall fix it to the edge of the divided mesocolon permanently with three or four tacking stitches.

Thus we close the lesser peritoneal cavity at once and therefore the patient cannot have a subsequent hernia of the intestine into the lesser cavity, which will interfere with the line of union. Now we are ready to clamp this portion of the intestines (the jejunum).

The doctor [Dr. Murphy's assistant] holds up the stomach and we get ready to put this clamp on the line of union, and so press the gastric contents out of this portion of the stomach. Now, then, we shall bring up again the ligament of Treitz. We desire particularly a no-loop operation. We are right at the duodenojejunal angle. This clamp is very light and elastic and does not need covering. Putting it on in this way, we now are ready to proceed with the approximation. The method we now use more than any other is a return to the oblong button and the outside tacking stitch. The outside tacking stitch consists—look!—in picking up the fibrous and serous layer of the stomach and the fibrous and serous layer of the jejunum in this manner. It is a running stitch, merely fixing these to each other. It is taken about one-third of an inch from where the button comes. Now we are ready to put in the puckering string for the button. We used to put in an interrupted stitch, but we now like the running stitch better. It gives a firm approximation all the way and is put in in a few seconds. Now, then, we are ready to insert the button. We first put it in on the intestinal side. Look! that is about one-half inch from the line of suture. I put the needle through all the coats of the bowel, taking four or five stitches, depending on the length of each. Now, then, the second needle is put in in exactly the same way, about one-eighth inch from the first and parallel to it. In that way I pick up all the coats again. While both needles are in in this way, I divide the intestine between them. Now we shall draw both these needles through. Now we insert the

button. Have your forceps on one end, so the bowel will slide over its opposite half. Changing your hold on the button so as to catch the cylinder, you get ready to tie the stitches which will hold this end. Put the knot always on the end of your button in this way, so when you cut the thread the button does not slip out, as Zellovitch's did in one of his early cases.

Now we are ready for the insertion of the other thread. We pick up this stomach-wall and go through all the coats with the sutures in the same way we put in the jejunal suture.

The mortality of the present day gastro-enterostomy is chiefly from pneumonia and, less frequently, from hemorrhage.

Now we shall put in the other half of the button. You see we control hemorrhage completely with the button, because of its elastic pressure, and we have lessened the tendency to pneumonia, we believe, by cutting down the duration of operation and anesthesia. Now we unlock the forceps, press the two halves of the button together, and take off the clamp. Now we continue this running stitch. We pick up the intestine again, and run the puckering string on the anterior just as on the posterior side of the button, carrying it over here from side to side and picking up the peritoneum in exactly the same manner as previously. That, you see, is giving a good line of approximation.

Now we return these viscera to the abdomen and let the omentum also slip in. We have restored these structures to about their normal position. There is the round ligament, which you see I am holding in my forceps. We unite the two peritoneal layers of the greater omentum by overlapping, so that we cover that abraded surface at the line of union.

Now we draw out the pyloric zone. We free the lower margin on the pancreatic side in order to get completely around the pylorus, just at the pyloric line. See! There is the pyloric vessel. We pass a forceps between these vessels and behind the pylorus, carefully spreading our way. There is a relatively avascular zone here, if you can only get to it. Remember how Pratt brought out the importance of operating through avascular zones when, many years ago, he took out the uterus without putting on a ligature, because he worked through an avascular

zone. You see it is just at the pyloric ring that we wish to put the constriction. You can go higher up and get around the stomach without any trouble; but in attacking this pyloric zone, we are at the place where it is the most difficult to get through. Now there is the ulcer shining through behind the pylorus. Such an ulcer forms a cicatricial band which gradually occludes the pylorus. The ligamentum teres is now divided from its fixation to the abdominal wall at the umbilicus. The peritoneum on it is split clear back to the commissure of the liver. You see how easily the ligament slips out of its peritoneal sheath. It is now exposed and freed for a distance of about four inches. The free end is grasped by the forceps which we had placed behind the pylorus, and the ligament is drawn through so as completely to encircle the pyloric zone. It is then united by an overlapping lateral suture and it firmly occludes the pylorus and supports the pyloric zone of the stomach in an elevated position. (See CLINICS.) We shall tie all these bleeding points and let the pylorus fall back to its normal position. As to the methods of occlusion and closure of the pylorus, the original plan was that of Mikulicz, the division of the pylorus with the knife. The results you might call very good with a *few* cases; but to get a truer conception of the danger involved one has only to study the records of Hofmeister, of Stuttgart, who had 25 consecutive cases of recovery after division of the stomach and closure of the pylorus and then had four consecutive deaths. Think of that! That is what appalls one in the open division of the stomach after the plan of Mikulicz. When one man has four consecutive deaths after presumably having mastered the technic, that causes every one to hesitate before dividing the stomach as a means of closing the pylorus.

Now we sponge dry and look over our field. There is the pylorus. I am fastening the side of the round ligament to the outer coats of the pylorus directly at the avascular pyloric line, much as one unites the tendo Achillis laterally. That secures the pyloric zone so it cannot escape from the point of constriction. Now I think you can all see the band as it is applied. That will become ultimately a cicatricial band tying

off the pylorus. That anchors the pylorus to the band. See! there is not the slightest oozing from the line of suture; it is perfectly dry and secure. That, we believe, is a better method than the actual division of the pylorus or the use of the omentum, whereby the omentum is put around the pylorus exactly as was the round ligament here. Of course, one has to make a much larger opening behind the pylorus in using omentum, but by getting into an avascular zone one is able to do it fairly well. Here we are uniting the parietal peritoneum. We have covered the exposed raw surfaces.

Let the record show that there was evidence of a very extensive pericholecystitis with a cicatricial fixation of the gall-bladder to the duodenum; whether the primary lesion was in the duodenum could not be determined, except that, from appearances, we believe there was a duodenal ulcer; that there was also a small penetrating ulcer on the lesser curvature of the stomach close to the pylorus, about three-fourths of an inch away; that this apparently was where the bleeding came from; that there was back of this ulcer, as a guide to its location, which otherwise I might have overlooked, a greatly enlarged gland; the gland, in fact, kept me persistingly looking for a lesion in that place.

This type of ulcer is probably as common as the large round type. It is, however, a much more serious ulcer from the standpoint of hemorrhage.

Let the record show that a posterior, no-loop gastro-enterostomy with the oblong button was performed; that after doing the posterior gastro-enterostomy the round ligament of the liver was freed from its attachment to the umbilicus and was wound around the pylorus and secured at that position by suture to the pylorus; that the gastro-enterostomy was performed with a running stitch of Pagenstecher linen outside the button and clear around it.

This is the most annoying type of ulcer we have from the standpoint of hemorrhage, and this "tap-root" type, as we are wont to call it, more frequently causes perforation than the large round type. The line of perforation of such an ulcer often leads directly toward a vessel, and this fact lends support to the

belief that the origin of such ulcers may be embolic. This temperature elevation, which is often associated with hemorrhage from the stomach, has an analogue in the temperature elevation which is associated with hemorrhage from the lung. Preceding such hemorrhage you nearly always have some elevation of temperature.

We are washing off the wound with lysol. When we open the alimentary tract we always use lysol as a disinfecting solution on the surface of the fatty tissue. Remember, your infections do not occur inside the abdomen, when you have opened the alimentary tract, so much as they occur in the fatty tissue of the abdominal wall. If you get colon bacilli in the fatty tissue, then you have trouble. The colon bacilli are not commonly present so high up in the tract, but they are present often enough on these surfaces to cause us to take this additional precaution. Lysol has a particularly destructive effect on the colon bacillus.

The method of closure of the pylorus in this case, as you saw, was by freeing the round ligament of the liver from its attachment to the umbilicus, and dissecting it back for a distance of $3\frac{1}{2}$ inches. Then the pyloric zone of the stomach was elevated and we endeavored to find a free, avascular zone which lies just where the vessels divide on the surface. That is the place where you may most easily escape injury to the vessels, keeping lateral to the pancreas for the entire circumference of the pylorus. Remember, the lesser peritoneal cavity does not come down so far as that. Finally, we succeeded in making an opening between the two structures and in passing forceps over and grasping the freed end of the round ligament. We drew the ligament around the pylorus, a little to the duodenal side, and sutured it so as completely to occlude the pylorus with this white, fibrous, ligamentous mass. When that step was completed, then we tacked the ligament to the pyloric zone to hold it in place. Then an anchor stitch was placed so the ligament could not slip down and interfere with the ducts below, or slip up and interfere with the vascular supply of the stomach. It will stay just where it was put.

This is just the type of ulcer which one would expect to find in this particular case, considering the hemorrhage as due to a gastric ulcer. This ulcer leads to much the same type of hemorrhage as occurs in the duodenal ulcer. A small ulcerating area penetrates down to a blood-vessel which, when eroded, bleeds back into the stomach. But in this case the symptoms were all gastric, showing the ulcer to lie on the gastric side of the pylorus. The abdomen is closed in the usual way.

[*Note.*—The wound healed by primary union. The patient went on to a complete recovery without any untoward events. She passed the button on the seventeenth day after the operation. She left the hospital January 6, 1914. She writes us, under date of April 10, 1914: "I am happy to tell you that I am able to resume my duties (as surgical nurse). My stomach is greatly improved, for which I am very thankful."—ED.]



SARCOMA OF THE OVARY WITH ROTATION OF THE PEDICLE. DIFFERENTIAL DIAGNOSIS. OPERATION

HISTORY

THE patient is a young unmarried woman, aged twenty years. The family history is negative. Her previous history is negative, except for a fall she had when twelve years of age, striking her back in the lumbar region on the edge of a step. She was in bed two days and the soreness soon left her.

Menstruation began at thirteen years of age, was regular and of the twenty-eight-day type, lasting four days, the patient using from four to five napkins daily. The last menstruation occurred a week ago and was normal.

During the month of February, 1913, she says that she noticed difficulty in starting urination and that the urine burned her. Three days later she had severe, cramp-like pains, beginning in the median line of the abdomen, a little above the umbilicus, and radiating down the left side and into the left thigh. The left thigh would often cramp at night. This trouble lasted only two weeks, and all the symptoms had completely subsided at the end of one month. She says she never vomited or passed blood in the urine at any time during this sickness.

She was perfectly well until January, 1914, when she was taken sick with severe, cramp-like pains in the right iliac region while at work, and had to go to bed. She did not become nauseated and did not vomit, and the next morning she felt perfectly well again.

About the first week in February, 1914, she noticed that her abdomen below the umbilicus was becoming large, but she had no pain. Her abdomen continued to increase in size, but she was able to wear corsets until Wednesday, April 1, 1914. Since that date the tumor has increased in size at least 25 per cent.

She has not been constipated. She has only a slight ache in the small of her back, and a dull, bearing-down sensation in her abdomen. She has never had any abnormal flow of blood from the vagina, has passed no blood in the urine or stools, and has had no vaginal discharge.

EXAMINATION.—The tumor extends from the outer margin of the iliac region on the right side to the same point on the left side, and from the pubis to just above the umbilicus, being slightly higher on the left side, where it extends to within one inch of the costal margin. The long axis of the tumor is from side to side. The tumor is extremely hard and somewhat lobular, flat on percussion, is not attached to the anterior abdominal wall, and is only slightly movable from side to side. Since January, 1914, she has lost considerable weight, but does not know exactly how much.

COMMENTS AND OPERATION

DR. MURPHY: A hydronephrosis can cause a large tumor in this particular place, but a hydronephrosis would not have compressed the neck of the bladder eleven months ago. I remember a case of Dr. Edebohls'. The patient had a tumor in the midline and the diagnosis made was ovarian cyst. On opening the abdomen Dr. Edebohls found the tumor to be a cyst of the kidney, displaced downward and resting over the promontory of the sacrum. You will note in the charts which I have had displayed for you on the wall that the kidney can be displaced downward to the brim of the pelvis, or may be fixed in the midline. You will find a case recorded in one of my published papers where the kidney rested in the midline, and we removed the stone from the kidney by the transperitoneal route, through an abdominal midline incision. With such a condition, however, she should have no pressure on the bladder. She has granular casts and leucocytes in the urine, but no blood.

It is important to note that two months ago the patient began to have such severe pain in the side that she took some brandy and water and went to bed, but that after sleeping all night she felt all right the next day. What occurs in connection with



Fig. 196.—Large round-cell alveolar sarcoma of left ovary.

tumors of the pelvis which can give you such sudden pain? Rotation of the pedicle is the most common occurrence. Thrombosis and rupture of one of the vessels in the cyst are also very common accidents. When there is only a 180 degree rotation, these pains are not present. When there is a 240 degree rotation, then the patients develop thrombosis of the vessels of the pedicle, and then comes the sudden enlargement of the tumor, which is what the patient says occurred here. From the time she felt that pain the growth of the tumor has been rapid. That rapid increase in size is exactly what occurs in strangulation of the pedicle or in rotation of the pedicle without a strangulation, but with venous thrombosis. The soreness has all disappeared and also the tenderness. The question is, what is the matter with her? First, is she merely pregnant? Second, is the condition a tumor of the uterus? Third, is it a tumor of the ovary? Fourth, is it a tumor of the kidney? Fifth, is it a tumor connected with the mesentery in the pelvis? Sixth, has she a cyst of the mesentery? Seventh, is it a tumor connected with the chyle circulation? Eighth, is it a bladder lesion, and, if so, is it a tumor of the bladder?

The menstruation is regular. I have not made a vaginal examination as yet. While I am examining her I want the doctor to read the history of another case that is now in the hospital, to contrast the history of this woman with the other history, and to see how important it is to listen carefully to all the details of a history.

[Intern reads histories of the present patient and also of case on page 445 of the present CLINICS.]

DR. MURPHY: I will give you the vaginal findings. The size of the uterus is small. It is displaced clear across to the left. The fundus is pushed over in an anteflexed position and is small. I cannot feel the right ovary anywhere. Extending high up in the pelvis and filling the floor of the pelvis all the way across is a firm, smooth, hard mass. There is no apparent depression into the vagina of the right fornix. There is some bulging of the left fornix, because the uterus has been pushed downward by the tumor. The uterus is not enlarged, but rather is smaller than

normal and is displaced downward and forward. This mass has had its origin in the pelvis. Let us ask her another question. When you had this pain in January, did the tumor get larger the next day?

PATIENT: I did not notice.

DR. MURPHY: I should like to know if that was the time the tumor escaped from the pelvis. This tumor, as it is now, could not affect urination. It seems that the disturbance in urination must have been due to the pressure of the tumor before its escape out of the pelvis. The tumor is now entirely out of the pelvis. The reason we presume the tumor was originally in the pelvis was the difficulty in urination, for which her doctor did not determine the etiology.

Now, contrast this history with the history of the other case which the doctor has just read. What is the matter with the second patient? Her pain was very acute and colicky in character with the onset of the flow. The doctor said there was an exudate and some clots. The adnexa were not palpable. Her temperature went to 101° F. yesterday, but is normal today. She has a leukocyte count of 14,000. It is an interesting question, why she stopped menstruating. She has been married a year. The doctor, in examining her in August, found a double vagina. She stopped menstruating in January. She began menstruating suddenly with pain on last Sunday, and passed some large clots. There was no fetus in the mass of clots, but there was decidua. The doctor said positively there was decidua, so I presume that he examined the mass microscopically. The cessation of menstruation, some enlargement of the uterus, a little fluid in the breasts, the sudden onset of pain at the end of practically three months of amenorrhea, no pains in the interim, indicate pregnancy. If she had a tubal pregnancy, she should have had the pain along about the fifth to the seventh week. If that was a decidua which she passed, she must have a pregnancy some place. She has a tumor which has grown rapidly in the last two months, but was not noticed until two months ago. The tumor is woody, hard, impacted in the pelvis, filling all the upper portion of the

pelvis. The cervix is at the lower portion of the tumor, and is soft, movable, and somewhat enlarged. The cervix is closed. She had no tumor that she knows of before she ceased menstruating. Pregnancy has been known to go on outside of the uterus, even to term, but pregnancy would not give you the firm, hard mass down in the pelvis. The tumor and the uterus form one mass, extending half-way to the umbilicus, and we are unable to get past the firm mass blocking the pelvis to feel the fornices. You have to think of a fibroid also, and that is why it is necessary to know about how she was flowing previous to the cessation of menstruation, three months ago. The question which comes up with this solid mass is whether she has a fibroid only or a pregnancy plus a fibroid. The doctor, in doing the operation last October, did not determine whether she had a double uterus. In my examination I could find only one cervix. She may have a double uterus and only one cervix. If the previous surgeon's statement is correct, she may have an impregnation in one cornu of her uterus. Such patients often have only one cervix, but two vaginas. They often have also a bicornute uterus, the one cornu of which may become impregnated and go on to full term, while the other develops only a decidua. She has a fibroid and, I believe, in addition to the fibroid, a pregnancy. That is the only diagnosis I could make which will fit the complicated conditions presented here. How could we verify the diagnosis of pregnancy? By the Abderhalden test. The flowing has all stopped now, and she is returning to her normal condition. Pulse and temperature are about normal, and it looks, indeed, as if the pregnancy were either in the tube or in the cornu of the uterus, or as if the fetus has escaped.

Now, what shall we do with this patient we have here on the table? We have made a diagnosis of a tumor outside of the uterus with the uterus anteflexed. The uterus is not a part of the tumor at all. The uterus is displaced to the left side. The tumor most probably is one of the ovary. It is not a par-ovarian tumor nor a simple cyst of the ovary or of the broad ligament. Why? Cysts of the broad ligament always bulge into the vagina, fluctuating even when under high tension. This

tumor is solid and has solid nodules on its outside. It is probably a solid tumor of the ovary. Did the sudden enlargement of which the patient tells us come from a hemorrhage or from a rotation of the tumor on its pedicle, or from its taking on a malignant character? Possibly this tumor may be a sarcoma of the ovary, which is one of the neoplasms that may occur at this age, and one of the conditions you should keep in mind as being most probable. Therefore, you have to advise that this woman be operated. We do not believe that this is a kidney tumor. We believe it is a tumor which originated in the pelvis and rose out of the pelvis probably when she had the severe pain she told us of, and we are very fearful that it is a malignant lesion.

[Incision. Abdomen opened. Tumor exposed.]

There is no doubt now about the tumor being malignant, judging from its appearances. A bloody fluid is present in the abdomen, a condition always very suggestive of malignancy. We shall endeavor to take out the tumor, but without much hope of curing her permanently. We operated on one woman with a sarcoma of the ovary four or five times and were able to prolong her life a number of years.

The omentum is firmly bound to something down here in the pelvis. It is probably infected, and I shall clamp it away up in here, as close to the colon as possible. Now I have freed and brought out the tumor and exposed its pedicle. That is very pretty—I mean the rotation of the pedicle which we can now see and which we spoke of as possibly the cause of her sudden pain before she came to the hospital. I said that I thought the rotation did not go far enough to strangulate the tumor. It went only two-fifths of the way, or it would have necrotized the tumor. For the individual satisfaction that it gives, I would rather have a good clinical history than a gold mine. I want to show you the twisted pedicle before I amputate it. You do not see such conditions often, nor do you see them often where they have been suspected and the probabilities analyzed as accurately as we were able to analyze this case.

There is the intestine—the large intestine. See the longitudinal bands on it! There is where the temptation is great to

go in and put the clamps on the pedicle, and, not recognizing the adjacent mesentery, get your clamp right onto it. There is the pedicle, with the two-fifths rotation still present. When you get more than three-fifths rotation there is strangulation, you remember. We shall swing that pedicle around, put on the clamp, and place our ligatures in the crease made by the clamp, thus making more direct compression on the vessels by the ligature than if a lot of connective tissue were also included in the ligature. Now we shall get these other adhesions to the tumor mass up in a position to tie them. See how the active growth of this neoplasm has produced a vascular supply to correspond to it! See the enormous vessels which have developed here in the adhesions! Here is some more of the bloody fluid that was in the abdomen. We shall bring up this portion of the freed intestine, of which we have the mesentery exposed, see how many bleeding points there are, and top-sew them. It is on the under side of the mesentery that these large vessels developed to form a collateral circulation in the tumor after the rotation occurred. I am infolding that portion of the intestine so as to cover the raw spots on the mesentery, so that there will be left no raw surfaces to which neighboring structures can become adherent. This is the wall of the tumor. There are some malignant foci in the omentum, so I have to cut it off above the point where the doctor first tied it, and excise it as close to the colon as possible. There are the subperitoneal lymphatics, which are all enlarged; but that fact does not necessarily mean that the growth is sarcomatous. Everything in the field is perfectly dry now. I am making an ectropion of the peritoneum in closing it, just as though I expected this woman to get well and stay well.

Let the record show that on opening the abdomen it was found to contain a bloody fluid, which we considered very suspicious of the lesion within being malignant—a malignant neoplasm at this age being more commonly a sarcoma. When you see a peritoneal fluid of this bloody character accompanying an abdominal tumor, the probability is 90 per cent. that the tumor is malignant. Occasionally with tuberculosis also you may have a

bloody fluid in the peritoneal cavity, accompanied by tumor formation.

Let the record further show that the omentum had become fully adherent all over the left side of the tumor, and was clamped, ligated, and divided high up toward the colon; that the tumor was firmly adherent to the under surface of the mesentery of the small intestine; that this adhesion was separated with great care without opening the intestine, and left a raw surface which was subsequently covered by an inversion suture; that the tumor was then elevated and was found to have made a two-fifths rotation of its pedicle, which corresponded with the clinical history of sudden pain and sudden increase in size of the tumor; that the tumor was also adherent over the left broad ligament, but passed otherwise freely over the peritoneum; that there were no adhesions to the caput coli; that the tumor originated on the left side, contrary to the patient's statement that she thought it came up first from the right side; that all the pedicles and adhesions were clamped with the angiotribe before they were tied, and that this method gave a good compression of the vessels; that there was no involution made of the stumps of the pedicles.

I want the doctor to read the history again.

[Intern reads history.]

There we get again the story of the rapidity of the growth, that is, that she apparently had the tumor eleven months ago, the only symptom then being difficulty with urination, so that she had to be catheterized every three days or so for about two months. She had no pain, but could not get the urine started. After each catheterization she would go about three days, urinating spontaneously, meaning that there was some outside obstruction to the emptying of the bladder. Therefore, she must have had the tumor there eleven months ago. Then she had no special symptoms until the rotation occurred, when she had the sudden pain, and following that the sudden enlargement of the abdomen, the tumor coming out of the pelvis and rotating as it came out. It did not rotate far enough to produce a complete necrosis. From the gross appearance of this tumor I should judge

that it is probably a sarcoma, possibly a carcinoma, but yet it looks more like a sarcoma.

What shall we do? As soon as she is over the effects of the operation we shall give her large doses of sodium cacodylate—five-, six-, seven-, eight-, or ten-grain doses at a single injection. In the cases in which we have given this drug our results have been excellent. You can see usually a pronounced diminution in the size of the tumor. We had one man here with multiple sarcomata in whom the growths disappeared, leaving only a few spots. Now, just when we thought we had him practically cured, he commences to develop new foci. He is getting an entirely new crop. We do not recommend this drug as a cure, and we are not writing about it yet. For some of these cases we have used "606," and for some we have used neosalvarsan. We intend using the sodium cacodylate intravenously as soon as we can get some one to do the experimental work to prove its safety.

Pathologist's report (Dr. Zeit): The specimen removed at operation is a solid tumor of the ovary. Save for the cut through its pedicle, its surface is smooth and undisturbed, and there are no portions of adjacent structures adherent to it. The microscopic sections show it to be an alveolar, large, round-cell sarcoma.

[*Note.*—The wound healed by complete primary union. The patient left the hospital on April 30, 1914, apparently entirely recovered.—ED.]

ANKYLOSIS OF THE JAW

A series of pictures illustrating the steps in Dr. Murphy's operations for ankylosis of the jaw, cases of which have been reported already in the CLINICS.

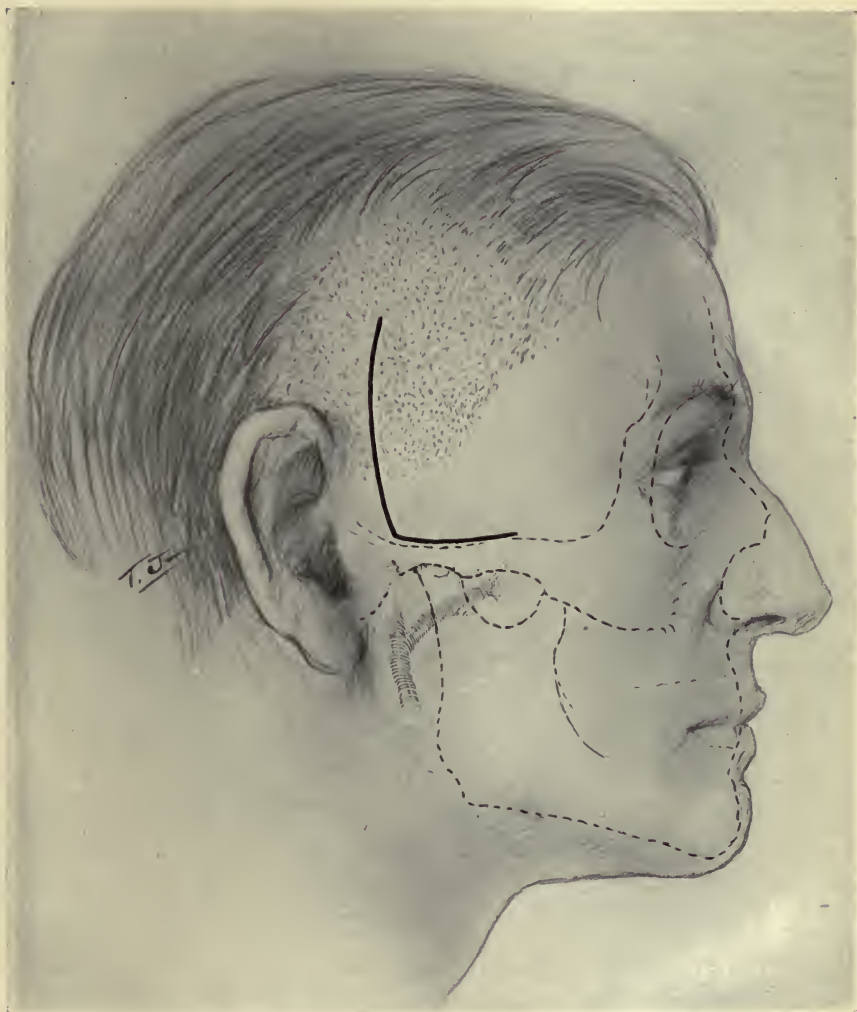


Fig. 197.—L-shaped skin incision above the zygoma and in front of the ear, so placed to avoid injury to the facial nerve. Note the relation of the external carotid, the temporal, and internal maxillary arteries to the field of operation. The last-named vessel in passing inward behind the neck of the mandible lies close to the bone and must be carefully protected from injury during the operation, especially at the time when the neck of the mandible is divided.

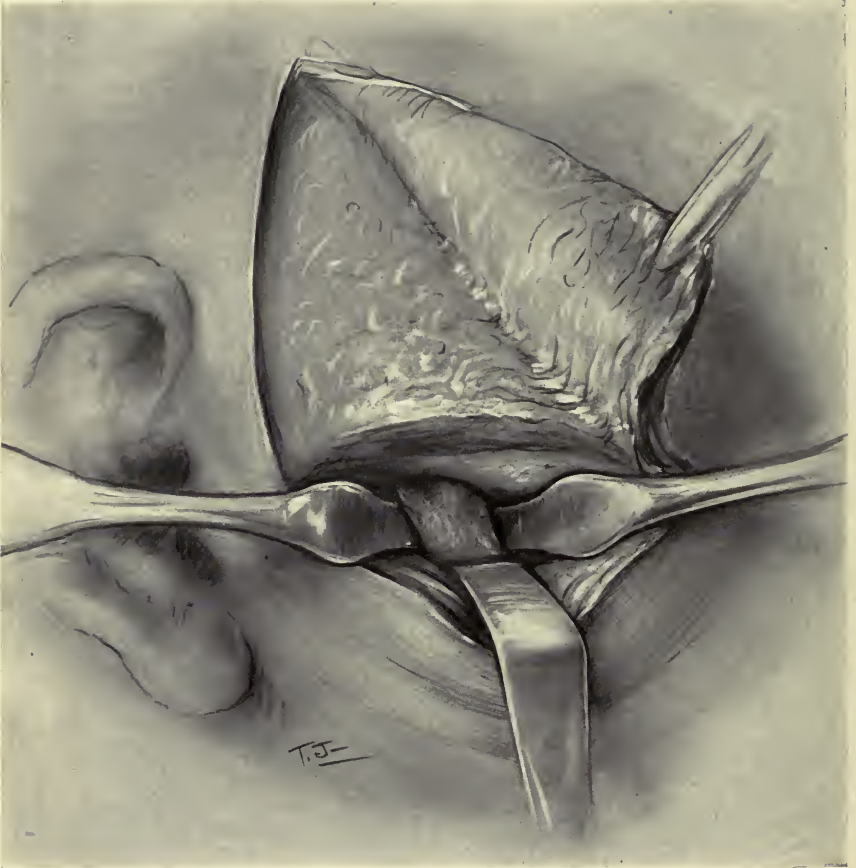


Fig. 198.—Skin-flap retracted and zygoma and neck of the mandible exposed. The two curved periosteal elevators are shown closely applied to the posterior surface of the neck of the mandible, thus protecting the internal maxillary artery from injury during division of the bone.

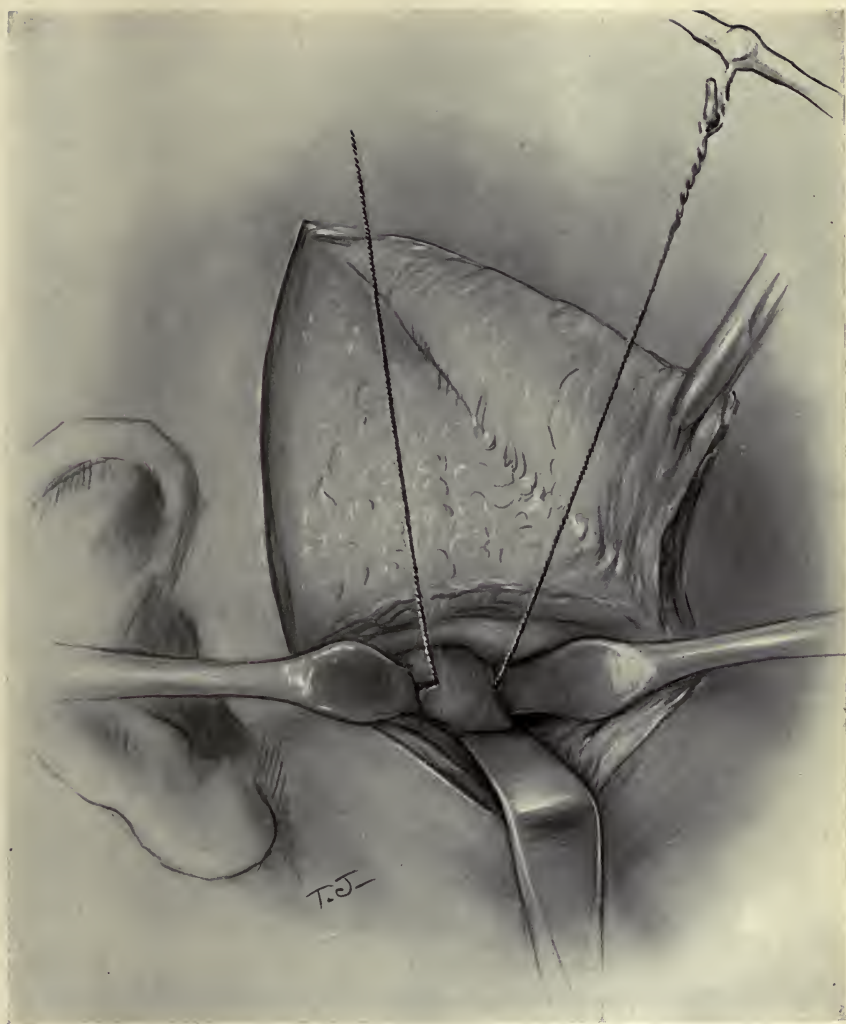


Fig. 199.—Dividing the neck of the mandible with the Gigli saw. (In actual operation the saw is not allowed to make so acute an angle as shown in the illustration, because of its great tendency to break when sharply bent.)



Fig. 200.—The neck of the mandible has been divided, the cut ends of the bone separated by traction on the lower fragment, and space thus provided for the interposing fascia and fat flap. The curved periosteal elevators still protect the internal maxillary artery from trauma.



Fig. 201.—The pedicled fascia and fat flap is dissected out from the temporal fascia, and the free end of the flap is turned inward between the divided ends of the mandible and sutured securely in place with tacking stitches.



Fig. 202.—The flap is now in place and the wound ready for closure.

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Clinics of John B. Murphy at Mercy Hospital, Chicago.
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